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STUDIES OF HUMAN FUNCTION AND EXPERIENCE WHILE AWAKE AND ASLEEP

by

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Submitted for the degree of Doctor of Science in the University of Edinburgh (old regulations).

1963

C O N T E N T S.

9. "Cortical function during human sleep".
In Ciba Foundation symposium The Nature of Sleep, 1961, pp. 343-348 (edited by G.E.W. Wolstenholme and M. O'Connor).
J. and A. Churchill Ltd., London.

10. "Falling asleep open-eyed during intense rhythmic stimulation".
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11. "On the origin of the EEG alpha rhythm".
Psychol. Rev., 1961, 68, 360-362.

12. "Induction of illusory and hallucinatory voices with considerations of behaviour therapy".
J. ment. Sci., 1962, 108, 196-212.

13. "The EEG, eye-movements and dreams of the blind".
Quart. J. exp. psychol., 1962, 14, 183-186.

14. "Effects of sleep deprivation on behaviour, subsequent sleep, and dreaming".
J. ment. Sci., 1962, 108, 457-465.

15. "Sleep mechanisms: recent advances".
Proc. roy. Soc. Med., 1962, 55, 910-912.

16. "Eye movements during active and passive dreams".
Science, 1962, 137, 601.

17. "Melancholia and barbiturates: a controlled EEG, body, and eye movement study of sleep".
Brit. J. Psychiat., 1963, 109, 66-78.

18. "Amphetamine and phenmetrazine addiction: physiological abnormalities in the abstinence syndrome".

Brit. med. J., 1963 (in press).

19. "Physiology of sleep accompanying dreaming".

In The Scientific Basis of Medicine Annual Reviews, 1962-63. Edited by Sir James Paterson Ross.

Athlone Press, University of London (in press).

Part III. (book, bound separately).

20. Sleeping and Waking: Physiology and Psychology. 1962. Elsevier, Amsterdam.

P R E F A C E.

The work described in the publications included in this submission did not contribute to the award of any other degree. Part I contains, in accordance with precedent, early works and is merely introductory. The substance of the present submission is to be found in Parts II and III. It should be pointed out that where, in Part III (my book), I allude to the references "Oswald, 1958, 1959b, 1959c, and 1959d", it is to work which formed the basis of my M.D. thesis of 1958, in which Fig. 24 of my book also appeared. It may, however, be added that the account given in the book of these particular studies is modified by subsequent work of which mention is sometimes made (e.g. p. 93).

In point of time the book falls somewhere prior to the work of publication no. 12 in Part II of the submission. The book is naturally a general review of the field and sets out my own interpretations and theoretical position. Taken as a whole, it does, however, contain a quantity of otherwise unpublished original material, illustrated, for example, in figs. 7, 8, 10, 12 and 32, and made up mainly of numerous minor points drawn from my research and personal experience (chapter 7 especially, perhaps).

There are two principal themes in the submitted works. Firstly, the study of human imagery (experiences of a sensory nature not based on reality) and, secondly, of human/

human sleep. The former might be said to have its roots in publication no. 3, with derivative studies nos. 1 and 2. In the course of the work of no. 1, an inexplicable phenomenon, namely, of rapid fluctuation of an after-image, gave rise to a case report (no. 4) and this puzzling phenomenon really led to all my subsequent work on sleep, for only in the neurophysiological basis of awareness could I find a plausible explanation, an explanation, I think, supported by the more recent work of Dr. E. W. Poole particularly (p. 27 of my book). Following this came EEG studies of very rapid fluctuation of cerebral vigilance (book, pp. 60-65). Publications nos. 5, 6 and 7 are outside the two main research themes.

In publications nos. 8 and 9, and in my book (p. 50, second para.), are described researches into the discriminative capacity of the human brain during sleep and the effects of meaningful stimuli. Inferences are made about the role of the cerebral cortex and this subject is taken further by studies of the effects of meaningful stimuli upon dream content (outlined in publication no. 19), most of the last work being done by a Ph.D. student of mine, R. J. Berger, though I played a part in the "blind" evaluation of the effects of such stimulation and in the background organisation of the research.

The sleep-promoting effect of rhythmic activity and stimulation has been a particular interest (no. 10). A

study/

study of a clinical disorder of sleep involving violent rhythmic activity (no. 19) forms a link with the purely experimental work.

Publication no. 11 is a minor one, making brief mention of some experiments. In publication no. 12 is an account, arising in a clinical context, of further studies of imagery with, I think it is fair to say, the first published "experimental" demonstration of frank auditory hallucinations of voices produced by psychological techniques. The role of the rhythm of stimulation is again stressed. General hallucinatory states, with systematic delusions in some cases, can be caused by sleep deprivation and instances are included in no. 14.

The original divergence of my research from the study of imagery to the neurophysiology of sleep has thus eventually led back to the study of imagery, not only in association with drowsiness (chapters 6 and 7 of the book) and sleep deprivation, but during dreaming. In publications nos. 13 and 15, research is described which would support the view that rapid eye-movements made during dreaming are related to the nature of the dream imagery.

A brief review of recent advances is found in no. 15 and a more extensive and more up to date review in no. 19, which also reports much original material not available elsewhere in the literature (exemplified in figs. 3-9). It brings my own theoretical position up to date, for in regard/

regard to the nature of the "paradoxical" or "hind-brain" phase of sleep my book is sadly and all too quickly out of date.

Two clinical studies of this latter kind of sleep are reported in nos. 17 and 18. Insomnia remains a baffling problem and the widespread use of hypnotic drugs calls for intensive study of their effects upon sleep. No. 18 reports a type of study of drug addiction of a new kind, namely, the demonstration of long-persisting abnormalities that can be measured during the period of the abstinence syndrome. It has some theoretical significance, for some American workers have written of "dream-deprivation", leading to a subsequent and consequent increase in the duration of "hind-brain" sleep. I prefer to consider deprivation of "hind-brain" sleep. The two viewpoints might be thought not mutually exclusive, the one being "psychological", the other "physiological". The patients who were fully addicted to amphetamine and similar drugs, while receiving those drugs showed no signs of being deprived of "hind-brain" sleep and indeed the values found were, on the whole, rather high. The huge increases on withdrawal could therefore not easily be explained on a psychological theory but offer no difficulty to a physiological theory.

Joint publications. The work submitted is a record

of/

of original, independent research in accordance with General Regulation III, with footnote, of the old regulations for the award of the D.Sc. degree. Publications nos. 8, 9, 13, 14, 16, 17 and 18 are joint publications with junior research associates.

A Ph.D. student of mine, R. J. Berger, made the greater contribution in originating and conducting the work of no. 14, though I did play a substantial practical role in it. Again, while I originated and largely conducted the analysis of data reported in no. 16, the material used had been collected by Berger in the course of his research towards his Ph.D. thesis.

In all the remaining joint publications, I was responsible for originating and organising the research and in each case bore the greatest single load of practical work as well as preparing the material for publication, the others taking part as assistants mostly without previous experience in the field. The patients used in the work of nos. 17 and 18 were all under my immediate clinical care. The length of no. 13 bears no relation to the work conducted by myself in seeking out from a variety of sources men who were not merely registered "blind" but totally blind, yet possessing two freely movable eyeballs and, above all, willing to be persuaded to go with a stranger wishing to conduct experiments while they slept in a mental hospital - a prospect too fearful for the majority of blind men.

PART I

Reprinted from THE QUARTERLY JOURNAL OF EXPERIMENTAL PSYCHOLOGY,
Vol. IX, Part 2, May, 1957.

AFTER-IMAGES FROM RETINA AND BRAIN

BY

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The evidence pointing to the retinal origin of after-images is considered. The reports of the occurrence of after-images from visual images of hallucinatory vividness are reviewed.

Experimental results are presented to indicate that a complementarily coloured after-image may arise following the exposure of the temporarily blind retina to a coloured stimulus.

After-images, or after-effects, from vivid images are described in seventeen persons (mostly possessors of "number-forms"). They are found to move with the eyes and to show, in some persons, a degree of conformity with Emmert's Law which, while considerable, is less than that of after-images of real stimuli. In the case of one "eidetic" subject, the after-images from neither real nor imaged stimuli conformed with Emmert's Law. In some persons, after-images of images occur in complementary colours.

The retinal origin of after-images is affirmed, but that they can occur occasionally as a purely central phenomenon is acknowledged. The possible learned or inherent nature of after-images of central origin is discussed.

I

INTRODUCTION

The problem of the retinal or central origin of visual after-images was for many years the subject of intense controversy. That after-images can be solely of peripheral origin has been shown by the fact that an after-image (hereinafter referred to as AI) may follow the exposure of a light-source to an eye which is temporarily blind, the blindness being caused by local pressure on the eyeball. The clearest demonstration of this is that of Cibis and Nothdurft (1948). The method had previously been applied to the same problem by Exner (1879) and Craik (1940). Cibis and Nothdurft covered one eye and made the other blind. The blind eye only was then exposed to an electric-light bulb, and later an AI was seen when the pressure was released. Their experiment did not relate to coloured light. Exner's (1879) experiments had included a coloured source and he failed to obtain AIs. Continental writers have emphasized the role of the cerebrum in colour-vision, describing cases of achromatic vision consequent on brain injuries. Among observations which are to be described below are some in which complementarily coloured AIs occur as central phenomena. It is therefore pertinent to consider the use of pressure-blindness to determine whether complementarily coloured AIs can have a retinal origin.

One source of evidence relating to AIs of purely central origin derives from the occurrence of AIs from hallucinated stimuli. This is a possibility that seems often to have been regarded as not quite respectable.

Gruithuisen (1812, p. 256) described how, on awakening from sleep, he experienced AIs from the visual phenomena of dreams. He experienced negative AIs ("umgekehrten Tauschungen") over a hundred times. He once had a positive AI. Dreaming of a lightning flash, he awoke, and observed an AI of "feeble light" which gradually faded to become darker than its surroundings. He also described how he dreamed of violet fluorspar on a fire of glowing coals, and on awakening, had an AI which moved with his direction of regard, and appeared as a yellow spot on a darker area, like the

sun in a stagnant pool (". . . die Gestalt der Sonne in einem stehenden Wasser hatte").

Alexander (1904) was to describe similar AIs of dreams. He mentions an example of a dream image of a human figure, "and when my eyes suddenly opened, I was surprised to see the figure lengthen out exactly as does an after-image."

Wundt (1863, p. 387) wrote that if a brightly-coloured image is regarded for a long time with eyes closed, on opening the eyes, and viewing a plain white surface, the image may persist briefly in its complementary colours ("so sieht man auf dieser kurze zeit das Phantasiebild in den Ergänzungsfarben fortbestehen"). The statement does not appear in the second edition, and may be among those "wild oats of my youthful days, which I would gladly have forgotten" (preface to the second edition, 1892).

The Salpêtrière workers, Binet, Féré and Charcot, were familiar with Wundt's view as quoted by Ribot (1882, p. 20), and with the fluorspar episode of Gruithuisen, as given by Burdach (1839, p. 207), the AI being described as a yellow spot "sur un fond bleu." Féré (1885) found he was able himself to obtain a green AI from a red imaged figure, but "très peu de sujets sont capables de la répéter."

It was found (Binet and Féré, 1898) that if they caused a coloured hallucination to be perceived by their hypnotic subjects, there followed an AI in the complementary colour.

Franz (1899) remained sceptical of the naïvety of the subjects despite the remark of Binet and Féré (1898, p. 253) that "it would be unreasonable to maintain that an hysterical woman, who scarcely knows how to read or write, knows the theory of complementary colours. Our subjects have always answered correctly and the correct answer has been given when the experiment was performed for the first time."

In St. Petersburg the same phenomenon was independently described by Sreznewsky and Bechterev (Bechterev, 1906) using hypnotized subjects.

William James (1890, p. 67) quoted the case of Meyer, who conjured up a vivid image of a stirrup with his eyes closed and, upon opening the eyes, "saw its after-image." Alexander (1904) pointed out that Meyer, in the passage quoted, does not state that he saw negative AIs as James assumes, and that there was no reason why the example given was not a projection of the original image. Alexander himself could still see images of chessmen with his eyes open an hour after playing chess, and their size was proportional to his distance from the background on which they were projected. He wrote, "some mental images follow precisely the law of after-images." He therefore disputed James' "universal proposition, that after-images seem larger if we project them on a distant screen, and smaller if we project them on a near one, whilst no such change takes place in mental pictures" (James, 1890, p. 51). He implicitly challenged Fechner's principle that such mental pictures do not move with the eyes.

Franz's remarks, in regard to the observation of Binet and Féré, led Downey (1901) to seek a totally naïve subject, and she gives an excellent account of a series of observations in which an image of a coloured form was followed consistently by a brief AI in the complementary colour. If Downey suggested to the subject that she should try and see a colour other than the complementary, there was delay while the subject first suppressed the spontaneous, complementary AI, and then imaged the suggested colour.

In studies of eidetic imagery, Jaensch (1930) found that some children could imagine a coloured object, then look at a screen and see the AI in a complementary colour. Klüver (1928) stated he had found the same phenomenon in eidetic subjects. Jaensch regarded it as a manifestation of extreme eidetic ability.

Dorcus (1937) carried out experiments on AIs of suggested colours with hypnotized subjects, but failed to obtain the older hypnotists' results. His hypnotic technique is not described and, in accord with the fashion of the day, he does not state if he obtained introspections from his subjects as to whether they had even experienced hallucinations. Since visual hallucinations are not the easiest hypnotic phenomena to obtain, this obviously bears on his results.

Erickson and Erickson (1938), using naïve subjects, confirmed the observations of the earlier hypnotists, that complementarily coloured AIs may follow hallucinated colours. They showed the same extraordinary patience and care in ensuring a profound trance that has marked much of Erickson's reported hypnotic work.

Hibler (1940) failed to confirm the Ericksons' results, but gives the impression that such was his intention when he embarked on his experiments. He did not use naïve subjects or a comparable technique (Erickson, 1941; Hibler, 1941), but obtained similar results. These results he rejected because when the subjects were rapidly awoken from the trance state the complementary AI no longer persisted. However, Downey, Weiskrantz (1950) and the writer's results reported below are in agreement that the spontaneous AIs from imaged stimuli generally last only about 5 seconds. In the hypnotic state it might be argued that the AI would receive its hue spontaneously, but because the subjects believed there to exist a genuine coloured piece of paper, would persist as an active haullucination.

Weiskrantz (1950) related the size of AIs from an imaged stimulus to Emmert's Law. He described the case of a woman (who was not naïve) who obtained negative AIs from an imaged black square. No chromatic AIs could be obtained. The sizes of the AIs corresponded closely with those produced by a real stimulus and those predicted by Emmert's Law.

Emmert's Law states that the linear size of an AI varies approximately linearly with the distance of the surface on which it is projected from the observer. $\frac{D_1}{D_2} = \frac{S}{I}$ (S and I being the linear size of stimulus and AI respectively; D_1 and D_2 being distance from observer to stimulus, and observer to surface on which the AI is seen, respectively), or $\frac{D_1}{D_2} \cdot \frac{I}{S} = K_E$. Calculation of K_E provides a convenient means of determining the degree of conformity of an image size with Emmert's Law (for complete conformity K_E being equal to unity). This function K_E is therefore used in all the tables of measurements recorded below.

II

Experimental Method and Results

(*a*) It was confirmed that, using one eye only, an AI may follow exposure of an electric-light bulb to the pressure-blind eye. The method was then applied to a coloured stimulus. A rectangular strip of red paper about 6 inches by 12 inches, of good saturation, was placed by an assistant about 2 feet in front of the already blinded eye of the writer in a previously agreed position on a plain white wall. "Fixation" of the position was then maintained for 20 seconds. The red paper was removed and the pressure released. With return of vision the wall was viewed for an AI. It had previously been determined that fixation of the paper for 20 seconds with a normal eye would give a bright green AI for 12 to 15 seconds, with the aid of blinking.

Peripheral, coloured AIs are not easily seen, and it was not till the ninth attempt that an unequivocal, bright green, rectangular and centrally-placed AI, which could be seen for about 15 seconds, was obtained. Evidently the correct spot had finally been fixated. After six more attempts another green AI was obtained.

(*b*) The subjects described below were mostly drawn from a larger group known o possess spontaneous vivid imagery. Fifteen of the total of 17 subjects possessed 'number-forms" or analogous visual imagery for months, days, alphabet or family, of the type first described and discussed by Galton and later by many others, including Pear (1922). The subjects, with one exception, were Royal Air Force personnel, and were all naïve at least in regard to Emmert's Law and the rules of complementary colours of AIs.

The first question put to each subject was "Can you close your eyes and see a red cross?" (or a black square). If the response was affirmative—"Keep looking at it for half a minute. I shall time you. When I ask you to open your eyes, I want you to do so and just look at the wall in front of you." The subject had been previously positioned in front of a plain wall (generally of a light buff colour). When the subject had opened his eyes, he was asked, "Did you see anything on the wall?" If the reply was affirmative, efforts were made to investigate the matter further, but this was not always possible. Many of the subjects were available only for an hour or two. All observations made on such subjects are recorded below without selection. Twenty possessors of number, or other, "forms" were questioned and fifteen found to experience after-effects of images.

On many of the subjects a series of measurements of the sizes of AIs were undertaken, using a method similar to that of Weiskrantz (1950). The subject fixedly regarded a faint pencil dot and imaged a square, the corners of which were given by four very small, faint dots on a large sheet of off-white paper, there being a light and fairly uniform background. When a square could be perceived, fixation of it was maintained for half a minute, after which the subject looked at a central spot on a very large sheet of off-white graph paper, the illumination of which was only very slightly greater than that of the stimulus paper. His instructions guided the experimenter's pointers on the graph paper, to obtain the size of the AI. Control experiments showed that the faint dots on the stimulus paper were incapable of producing AIs. The dots formed a 2-inch square in the majority of cases, but occasionally other sizes were used. The size was not known by the subject. Similar observations with real stimuli were also carried out. The distances from the subjects' eyelids to the stimulus, and from the subjects' eyelids to the screen, were measured in every observation by tape measure.

Subject Ho.

A twenty-six-year-old medical officer, a colleague of the writer's, working as a plastic surgeon. He imaged a red cross with eyes closed and saw a large, dark green cross on eye-opening. This was repeated. A violet square became a yellow square on eye-opening. The imaged violet square had had a yellow edge around it. An image of an orange became a yellow-green on eye-opening and flashed to sky-blue when he blinked. A green square became red. Blue became a lime-green.

The subject remarked, "If I look at a light colour, when I open my eyes it reverses to dark and vice versa. I seem to see green easily. Is it because when you look away from a bright electric light you see a green horseshoe?"

At this point, and subsequent to the above observations, he was questioned in regard to his preconceived ideas, if any. It may be pointed out that a knowledge of the phenomena of AIs is not required of, nor possessed by, the majority of medical students or practitioners. He proved remarkably naïve and denied any knowledge of the rules of complementary colours. His only suggestion of mechanism was to repeat his remark about the electric-light bulb.

He was asked to try and see a colour other than the complementary when he opened his eyes, but reported, "When I'm concentrating on a coloured object I can't think of anything else or the object goes. I'm too busy concentrating on it to think of anything else, and when I do open my eyes it's too quick—there's no time to think."

Having had the "practice" of evoking images he found (as did others) it became easy to evoke them with eyes open. He was quite intrigued with the phenomenon, and pursued it silently on his own. He reported that an image of an orange had a halo of green around it. When he stopped trying to see the orange he suddenly saw a green image which abruptly shrank. A red cross with eyes closed became green on eye-opening, merging into a green circle, the peripheral corners seeming to expand, the central corners to fill in. The primary images and the AIs moved with eye-movements.

Observations with Subject Ho, which were subjected to measurement, are shown ir Table I. In this and the other tables the size of the AI, as predicted from Emmert's Law is given for each observation, together with the function K_E.

TABLE I

(SUBJECT HO.)

Obsn.	D_1 (ins.)	D_2 (ins.)	Stimulus			AI		Emmert (ins.)	K_E
			Imaged	Real	Size (ins.)	(ins.)	Colour		
(a)	41	70	Black		2	6	Black	3·5	1·7
(b)	41	70	Light Green		2	8	Dark Green	3·5	2·3
(c)	24	68	Black		2	11	Bright	6	1·8
(d)	45	87	Dark Green		2	9	Yellow	4	2·2
(e)	32	90	Grey		2	17	Bright	6	2·8
(f)	32	90		Blue	2	7·5	Yellow	6	1·3
(g)	48	90		Grey	2	4	Bright	4	1·0

After observation (*d*) the subject remarked that it seemed that the size of the AI was proportional to distance, and he proposed the appropriate equation, of which he had no previous knowledge. It is nevertheless apparent that the AIs to imaged stimuli, while showing a general trend towards increase of size with increase of $\frac{D_2}{D_1}$, did not conform to Emmert's Law, unlike real stimuli.

This subject's AIs to imaged stimuli lasted up to 10 seconds.

TABLE II

(SUBJECT CH.)

Obsn.	D_1 (ins.)	D_2 (ins.)	Stimulus			AI		Emmert (ins.)	K_E
			Imaged	Real	Size (ins.)	(ins.)	Colour		
(a)	19	60	Grey		2	5	Grey	6·0	0·8
(b)	19	78	Grey		2	7	Grey	8·0	0·9
(c)	25	52	Grey		2	4	Grey	4·0	1·0
(d)	15	52	Grey		2	5	Grey	6·9	0·7
(e)	15	53		Grey	2	6	Bright	6·9	0·9
(f)	23	42	Grey		2	3·5	Grey	3·5	1·0
(g)	23	107	Grey		2	7·5	Grey	9·3	0·8
(h)	23	107	Dark Grey		2	9	Bright	9·3	1·0
(i)	23	78	Grey		2	nil	—	—	—
(j)	23	78	Grey		2	6	Grey	6·8	0·9
(k)	23	78		Grey	2	7	Bright	6·8	1·0
(l)	42	76	Grey		2	4·75	Grey	3·6	1·3
(m)	42	76		Grey	2	3	Bright	3·6	0·8
(n)	51	93	Grey		2	3·5	Grey	3·6	1·0
(o)	51	93		Grey	2	3·75	Bright	3·6	1·0
(p)	70	30	Grey		2	3	Grey	0·9	3·5
(q)	70	30		Grey	2	1·4	Bright	0·9	1·6
(r)	70	30	Grey		2	2·5	Grey	0·9	2·8

Subject Ch.

A building apprentice aged nineteen, National Service, grammar school education, chief interest handicrafts, negligible physics. Totally naïve in regard to AIs. At the end of the series of observations the size-distance relation was explained to him. He denied having realized the relation and seemed to have some difficulty in grasping it.

He could not image colour, but could image a grey square, which moved with shifting of gaze. On glancing away to the screen, the square persisted only for a couple of seconds, in a new size and generally as a grey square. The indication of size was obtained from what might be called the primary memory image of the square—no actual perception of a square being present at the time the pointers were finally positioned. This should be borne in mind in considering the results in Table II, which show a remarkable conformity to Emmert's Law. It will be noted that the relation breaks down, for imaged stimuli, when the ratio of D_1 to D_2 is greater than unity. After observation (*q*) with a real stimulus the subject remarked that the AI was much smaller than the previous one.

This subject cannot, except in observation (*h*), be said to have experienced AIs from imaged stimuli, but rather the persistence of the original image.

Subject Ha.

A nineteen-year-old airman of secondary modern school education. Questioning after the observations revealed him to be completely naïve in regard to the normal properties of AIs. He possessed a circular "month-form," an "alphabet-form" with a red "H," and the idea of Sunday or Tuesday was accompanied by a sensation of white or blue respectively. This varient of synaesthesia is quite common.

Questioning elicited:—(*a*) with eyes closed a black cross changing to a large white cross on looking at the wall, (*b*) a red cross changing to a large dark cross and shrinking to a ball on eye-opening, (*c*) a black cross which became a large black cross on eye-opening. The subject remarked spontaneously that there was a "brightness" round the edges of the black figure. (*d*) Again, later, a black cross, but this he perceived as a crucifix type with himself regarding it obliquely, looking from an angle along its long axis. The AI was black, but the "horizontal" arm of the cross was now the longest, while previously it had been subjectively the shorter arm.

TABLE III
(SUBJECT HA.)

Obsn.	D_1 (*ins.*)	D_2 (*ins.*)	*Stimulus*		*AI*		*Emmert* (*ins.*)	K_E
			Imaged	*Size* (*ins.*)	(*ins.*)	*Colour*		
(*a*)	125	40	Blue	6	2	Yellow	1·9	1·0
(*b*)	30	59	Black	2	4	Bright	4·0	1·0
(*c*)	16	34	Green	2	3 tilted	Green	4·0 + tilted	0·7
(*d*)	133	39	Blue	6	2	Light Green	1·8	1·1
(*e*)	41	118	Blue	2	5	Blue	5·8	0·9
(*f*)	22	64	White	2	3	Dark	6·0	0·5
(*g*)	22	64	Black	2	7·5 tilted	Black	8·0 tilted	1·0

Further observations with this subject are shown in Table III. Unfortunately, more were not possible as, at the time he was seen, he was going overseas.

Observations (*c*) and (*g*) were of especial interest. On glancing at the screen his square had tilted, slightly in (*c*) and through 45 degrees in (*g*).

Subject Wi

A senior N.C.O., aged 37, a trained mental nurse of average intelligence. A red cross with eyes closed became, on eye-opening, a large orange cross which passed through orange to green, the green becoming circular and shrinking.

It may be pointed out that the knowledge of psychology possessed by mental nurse does not normally extend to AIs. At the end of the observations described in Table IV the subject was questioned and denied having any idea of the purpose of the observations or having any knowledge of AIs, let alone Emmert's Law.

TABLE IV
(SUBJECT WI.)

Obsn.	D_1 (*ins.*)	D_2 (*ins.*)	*2-in. Stimulus*		*AI* (*ins.*)	*Emmert* (*ins*)	K_E	*Remarks.*
			Imaged	*Real*				
(*a*)	26	56	Black		5	4·3	1·2	Green, rapidly to white, slowly shrinking to small bright spot. 20 secs.
(*b*)	26	56	Yellow		4·75	4·3	1·1	Grey, shrinking—becoming bright blob. 20 secs.
(*c*)	26	56	Blue		4·5	4·3	1·0	Light blue to green, to yellow, to white spot. 20 secs.
(*d*)	26	56		Blue	8	4·3	1·9	Yellow, to 4 in. square, shrinking to small white circle. 30 secs.
(*e*)	26	56		Blue	6	4·3	1·4	Yellow, to 4 in. square, to white spot. 30 secs.
(*f*)	16	43	Black		4·25	5·4	0·8	White, shrinking. 20 secs.
(*g*)	16	43		Grey	4·75	5·4	0·9	White, shrinking. 20 secs.
(*h*)	24	82	Red		5·75	6·8	0·8	Cream, became white circle, shrinking. 45 secs.
(*i*)	24	82		Grey	6·5	6·8	1·0	White, became grey, disappeared expanding outwards. 15 secs.
(*j*)	23	70	Black		6	6·1	1·0	Light grey, became circular, shrinking. 30 secs.
(*k*)	23	70		Grey	6·25	6·1	1·0	White, shrinking. 30 secs.
(*l*)	35	67	Apple Green		4	3·8	1·0	Light blue, shrinking through white, circular. 20 secs.
(*m*)	35	67		Grey	4	3·8	1·0	White, shrinking, becoming circular. 20 secs.

This subject's AIs from imaged stimuli lasted much longer than other subjects', generally persisting 20–30 seconds. During this time they became circular, underwent shrinkage, colour changes, and often a curious sense of pulsation of size, as if getting slightly bigger and smaller. Investigation of the pulsation rate was subsequently carried out and will be described elsewhere. The rate was that of his arterial pulse. His AIs to real stimuli also showed pulsation, but this was much less marked.

Subject Gr.

A National Service Corporal, aged 22, a trained mental nurse. Subsidiary Higher School Certificate in Physics. Discussion, after the observations described below, revealed he previously realized the existence of AIs and was vaguely aware of their colour relations, though not of size relations. There was no reason to doubt his trustworthiness. He stated that he was at first "amazed" to see any AI as a result of imaging objects. By the end of the observations he realized the size changes might obey the rules governing cinema projection, but thought his AIs much too large.

He imaged a red cross with eyes closed. On eye-opening he saw a much bigger green "blob" which genuinely surprised and interested him. A mauve cross was succeeded by a large, yellow, vertical streak, lasting 15 seconds. A green rectangle was succeeded by a large red one. A yellow rectangle became grey. These AIs all moved with change of direction of gaze.

His detailed results are given in Table V. Observations (*a*) to (*e*) were made on one day and show a remarkable conformity with Emmert's Law. The remaining observations

were made a month later. After observation (*p*) the subject remarked, "That seems too big, doesn't it?" The AI in (*r*) underwent growth upwards by about an inch after the first second or so. His AIs from imaged stimuli lasted 5–10 seconds.

TABLE V
(SUBJECT GR.)

Obsn.	D_1 (ins.)	D_2 (ins.)	Stimulus			AI		Emmert (ins.)	K_E
			Imaged	Real	Size (ins.)	(ins.)	Colour		
(*a*)	15	49	Black		2	5	White	6·5	0·8
(*b*)	15	81	Black		2	11	White	11	1·0
(*c*)	15	59	Black		2	8	White	8	1·0
(*d*)	14	32	White		2	4·5	Dark	4·6	1·0
(*e*)	140	42	Black		6	1·75	White	1·7	1·0
(*f*)	80	100	Weak Orange		6·6	8·5	Grey	8·2	1·0
(*g*)	80	100	Blue		2	5·5	Pink	2·5	2·2
(*h*)	80	100		Grey	2	3	White	2·5	1·2
(*i*)	50	82	Red		2	nil	nil	—	—
(*j*)	50	82	Black		2	4	White	3·3	1·2
(*k*)	50	82	Black		2	4·5	White	3·3	1·4
(*l*)	50	82		Grey	2	3	White	3·3	0·9
(*m*)	63	28	Black		2	1·2	Pink	0·9	1·3
(*n*)	95	34	Black		2	1·4	Yellow White	0·7	2·0
(*o*)	95	34		Grey	2	0·8	Yellow	0·7	1·1
(*p*)	34	108	Green		2	7	Bright	6·4	1·1
(*q*)	22	110	Dark Purple		2	12	White	10	1·2
(*r*)	77	105	White		2	4	Grey	2·7	1·5
(*s*)	77	105	Black		2	7	Bright Pink	2·7	2·6
(*t*)	77	105		Black	2	3	Yellow	2·7	1·1

Subject Wh.

A Sergeant, a trained mental nurse, aged 29. He claimed to be naïve in regard to the whole subject. He possessed visual imagery of hallucinatory vividness, which conformed with those criteria which were formerly considered to indicate "eidetic" ability. He could regard an object briefly, then turn away and see it wherever he wished, the image being "literally seen" in its original colours, though he was aware of it not being real. He experienced colour sensations with emotions.

It may be noted that the AIs of real stimuli, perceived by eidetic subjects, have been noted elsewhere as deviating markedly from Emmert's Law (Allport, 1924; Meenes and Morton, 1936).

Initial questioning elicited a clear red cross with eyes closed, which became a huge red cross on eye-opening. His detailed results are given in Table VI. His AIs from imaged stimuli lasted up to 3 or 4 seconds and moved with the eyes. He was asked, as previous subjects had been, to indicate the height of the AI. He complied with the request and until observation (*k*) it never occurred to him to remark on the width. In observation (*k*), fixation of a real blue 2-inch square was followed by a yellow AI, which he sketched for me, extending 4 inches below and 10 inches above the fixation point. Questioned, he stated calmly that it had behaved in the same fashion as all the previous AIs, the upper half giving the impression of elongating upwards. Asked why he had not mentioned this before, he replied that he had not been asked for anything except the height, and that he had assumed the elongation to be probably part of the natural response being studied. How great a part the experimenter's interest in the height had influenced the AIs up to this point, can only be surmized. Certainly they show a remarkably constant tendency to be about twice the expected height.

The colour of the AI in observation (*a*) was not recorded immediately, attention being directed to size; it was considered dark in retrospect. After the green AI in (*b*) the subject spontaneously expressed himself surprised, and asked why it should be green, when he

TABLE VI

(SUBJECT WH.)

Obsn.	D_1 (ins.)	D_2 (ins.)	Stimulus			AI			Emmert (ins.)	K_E
			Imaged	Real	Size (ins.)	Colour	Height (ins.)	Width (ins.)		
(*a*)	23	60	Red		2	Dark	11	–	5·2	2·1
(*b*)	23	60	Red		2	Green	10·5	–	5·2	2·0
(*c*)	23	60	Orange		2	Light Green	10·5	–	5·2	2·0
(*d*)	20	40	Black		2	Black	6·5	–	4	1·6
(*e*)	20	40	Yellow		2	Green	6	–	4	1·5
(*f*)	22	81	Green		2	Green	16·5	–	7·4	2·2
(*g*)	22	81	Violet		2	Violet	14	–	7·4	1·9
(*h*)	128	40	Black		6	Black	4	–	1·9	2·1
(*i*)	128	40	Black		6	Black	3·5	–	1·9	1·9
(*j*)	27	78	Green		2	Dark	9	–	5·8	1·6
(*k*)	27	78		Blue	2	Yellow	14	7	5·8	2·4 or 1·2
(*l*)	27	78		Blue	2	Yellow	14	7	5·8	2·4 or 1·2
(*m*)	27	78		Blue	2	Yellow	14	7	5·8	2·4 or 1·2
(*n*)	132	42	Black		6	Green	8		1·9	4·2
(*o*)	28	107	Black		2	Green	18	9	7·6	2·4 or 1·2
(*p*)	19	56	Green		2	Dark	12	6	5·9	2·0 or 1·0
(*q*)	19	40	Yellow		2	Red	7	3·75	4·2	1·7 or 0·9
(*r*)	19	40	Green		2	Dark	5	5	4·2	1·2
(*s*)	19	40		Grey	2	Green	6	6	4·2	1·4
(*t*)	31	120		Green	4	Green	11	–	15·5	0·7

had been seeing red. It will be noted that many of this subject's AIs are not complementarily coloured; green predominates. Observations (*a*) to (*i*) were made at the same session, (*j*) to (*n*) a week later, (*n*) being made some time after (*m*), the interval being occupied by considerations of the subject's edidetic images of real objects. These eidetic images were usually slightly larger than the real objects and showed no size-distance relation. It is possible that the size of the AI in (*n*) was influenced by the "set" derived from the eidetic images. Observations (*o*) to (*t*) were made a further week later. The subject remarked that the AI of observation (*r*) was the first one he had ever experienced as a square.

This subject's imagery was exceedingly labile and probably the AIs were greatly influenced by his attitude. Nevertheless, they appeared spontaneously and immediately on glancing at the screen, whereas the stimulus image, which was viewed for half a minute, always involved effort and delay before appearing.

Subject Ga.

An intelligent, nineteen-year-old airman, grammar school education (Arts subjects). Enquiry had previously elicited that he experienced different colour sensations when he thought of different days of the week. If he thought of Tuesday, for instance, with his eyes closed, he experienced a vivid sensation of green; with eyes open it was an "impression" only.

He was asked to visualize a red circle with his eyes closed and stare at it for a full minute; on eye-opening he saw a green circle on a grey screen in front of him. Asked to visualize blue, he said he saw a turquoise; on eye-opening this became a "sort of light green, not like the other green, this is a light colour." When asked to see a red circle and to try to go on seeing it on eye-opening he was able to do so. A green oval, visualized for 45 seconds with eyes closed, became a pink oval on the cream-coloured wall. Navy blue was succeeded by a "light colour." The subject remarked at this stage, that it seemed he was experiencing the complementary colour. He denied, on questioning, any previous knowledge of AIs, or that he had had any preconceived ideas as to what he would experience on opening his eyes.

He was asked to try to see yellow after visualizing red. He found, as did other subjects (and Downey's case) that he first had to suppress the spontaneous, green AI which appeared without effort. He could then perceive a yellow which grew more intense with time, whereas the spontaneous, effortless AI was at its most vivid initially.

His AIs from images moved with the eyes and lasted about 5 seconds. An image of a light bulb became a large dark shape, when he opened his eyes and regarded a distant wall.

Subject Hd.

A twenty-one-year-old airman, with "coloured months" and a "week-form." Wholly naïve in regard to AIs. He was genuinely surprised to see a large green square when he opened his eyes after imaging a red square.

Subject Bo.

A twenty-three-year-old housewife, who experienced colour sensations when she thought of certain months of the year, certain personalities or certain days of the week. The eyes being closed, the idea of Monday or Wednesday or Friday was accompanied by a blue or red or green sensation respectively.

She was able to visualize a black square on a large sheet of white paper on which were very faint dots forming a square 1·1 inches in height. This she regarded for 45 seconds at 22 inches from her eyes. On looking at the blank wall 19 feet away, she experienced a grey square, which faded in about 10 seconds. This AI was measured and found to be 12 inches in height. By Emmert's Law the predicted height of the AI would be 11·4 inches.

Unfortunately time did not allow further observations.

Subject Od.

A twenty-year-old airman. No "forms." Able with difficulty to visualize a red cross with eyes closed; saw a "mustard-coloured patch" on the buff wall on eye-opening.

Attempts to image a square with eyes open, on the stimulus paper used with other subjects, proved very difficult and he could do so only once. A 2-inch blue square became a "very vivid" blue square on the screen, tilted through 45 degrees (cp. Case *Ha*). This was of only 1·6 inches vertical height from corner to corner. $D_1 = 34$ inches, $D_2 = 55$ inches.

Subject N.

A twenty-four-year-old Corporal visualized a black cross with eyes closed. On eye-opening it was momentarily seen projected on the wall, turned through about 45 degrees.

Two other subjects repeatedly got large, bright, projected images from dark ones, on eye-opening, but could not image a square with eyes open. A pink square, with eyes closed, remained pink on eye-opening for one of these subjects.

Four other subjects were found, in whom the image of a black or coloured object briefly persisted unchanged on eye-opening (*cf.* Alexander's remarks on James' account of Meyer, and Subject Ch. above).

Weiskrantz (1950) used as a control a non-naïve subject familiar with Emmert's Law. His calculated sizes for AIs, when he sat in the experimental situation, deviated grossly from the sizes which should be predicted, and bore no comparison with the sizes of the genuine, spontaneous AIs from an imaged square, of Weiskrantz's case.

Similar control results were obtained by the writer. If, however, the control, a non-naïve subject, of higher educational level than most of the fifteen cases described above, was not required to fixate the imaginary square, but allowed to look where he liked, his estimates were considerably more accurate, though less than for the genuine AIs. His whole appearance was that of one making a shrewd guess—looking at the distance involved,

making slight movements of the head to judge the distance the better. A complete contrast to those who reported the presence of the image on fixation of the stimulus paper; they were concentrating with fixed gaze and, if they spoke, would often do so in a characteristic "careful not to distract" manner.

III

Discussion

The conclusion may be drawn from the observations described that AIs can occur as a purely central phenomenon. Filehne (1885) described how disappearance of an existing AI could be effected by means of pressure-blindness. Cibis and Nothdurft (1948), using a black and white figure as stimulus, studied this phenomenon and noted that they could still see an AI during a brief period, when awareness of the environment had just been caused to disappear by pressure on the eye. This AI they believed to be of wholly central origin. Central AIs are very individual matters and the writer, in the course of many experiments, has never observed an AI under the conditions described by Cibis and Nothdurft, unless some vision was still present. The writer's criterion of complete blindness was unawareness of a *moving* source of bright light. This is a more total form of blindness than mere unawareness of the static scene. On this account, and in view of McDougall's (1901) work on the unfluence of attention in causing awareness and fading of different parts of a visual field, Cibis and Nothdurft's belief that they were definitely seeing central AIs in all cases, should perhaps be regarded with caution.

It is pertinent to recall the local reduction of sensitivity in the visual field of one eye, following stimulation of the corresponding area of the other eye (Dunlap, 1921, Crook, 1930). This represents a form of central adaptation such as may accompany central AIs.

Scrutiny of the accounts of AIs following images or hallucinations as reported by others, and those here recorded, confirm that spontaneous AIs in complementary colours may occur. This colour relation, while a general finding, has exceptions, particularly if the eyes are open when the primary image or hallucination is perceived.

Alexander's (1904) doubts of James' "universal proposition" are confirmed; mental pictures can undergo size changes when projected on a distant screen and, as Alexander pointed out, those size changes frequently show conformation with Emmert's Law. The degree of conformation varies with different individuals, and is generally less exact than is the case with AIs of real objects. The "eidetic" subject's AIs from real and from imaged stimuli, deviated markedly from the sizes predicted by Emmert's Law.

Alexander's challenge of Fechner's widely quoted distinction between memory images and AIs, that only the latter move with the eyes, is supported.

The conformity of mental images with Emmert's Law demonstrates a cerebral correlate of angular size of a part of the whole visual field. This cerebral process persists in the absence of visual stimulation. The tentative conclusion might be drawn that the construction of a vivid image may involve many of the same neurones as are involved in the perception of a similar real stimulus.

The occurrence of a complementarily coloured and negative AI from an imaged stimulus might be learned or inherent.

If the response was a learned one it could be considered as a conditioned response, the unconditioned responses having occurred in past experience. Experimental "sensory conditioning" has always been difficult to achieve. That it can occur, if distractions are excluded, was shown by Leuba (1941) in a straightforward conditioning situation. Leuba obtained "limitation of the spontaneous mental life of the subject

and the consequent limitation of attention to the stimuli provided by the experimenter" by using hypnosis.

Popov (1953), under the relaxed, quiet and darkened conditions of electro-encephalographic recording, demonstrated that AI's can occur as conditioned responses and that the conditioned AI of a white light may show the same colour changes as the unconditioned AI response (Popov and Popov, 1953).

Alternatively, the complementary AI from a coloured image might result from central "fatigue," and be inherent in central colour perception mechanisms. McDougall carried out a series of experiments on the central inhibition of visual stimuli and demonstrated "monocular struggle," in which, under certain conditions, if light of two different colours falls on the same area of the retina of one eye, perception of first the one and then the other colour may alternate (McDougall, 1901, Observation 27). This experiment is easily carried out and has been confirmed by the writer using McDougall's method. McDougall's experiments led him to postulate a central origin for the "flight of colours" in the AI of a white light, and that central fatigue of one primary colour "system" may occur, with the appearance of another and inhibition of the first. An analogous fatigue process of one colour "system," and in consequence, a relatively active, unfatigued, alternative colour "system," might play a part in giving rise to AIs from images.

It may in conclusion be re-affirmed that perception of an AI normally arises in the presence of corresponding stimulation of nerve endings in the retina, but that in some persons it may occasionally arise solely as the result of cerebral activity. It is further believed that the nervous system will, at all levels, retinal and central, "register" a change of the input applied to it, and that this situation normally follows stimulation by light.

To all those who acted as subjects in these investigations, I wish to express my thanks. Also to my wife and Mr. P. Boss for assistance with translations. I am greatly indebted to Group Captain V. H. Tompkins, Principal Specialist in Neuropsychiatry, Royal Air Force, for his interest and encouragement.

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Reprinted from THE QUARTERLY JOURNAL OF EXPERIMENTAL PSYCHOLOGY,
VOL. IX, Part 3, August, 1957

THE E.E.G., VISUAL IMAGERY AND ATTENTION

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The claim that persons may be broadly classified into imaginal types on the basis of their E.E.G. records is examined. It is found that a given individual may show a different "type" of E.E.G. from one occasion to the next. A group of persons, known to indulge spontaneously in certain recognizable varieties of vivid visual imagery, contained the same proportion of the different E.E.G. "types" as a large control sample. It was found that subjects could experience changing or static visual images without any blocking of the alpha rhythm providing that difficulty was not experienced in perceiving the images. Flat E.E.G. records were not found to be associated with regular respiration.

It is proposed that the previously reported association between alpha blocking and the appearance of visual imagery could have arisen from the fact that difficulty in thinking (*a*) activates mechanisms which desynchronize E.E.G. potentials, (*b*) provokes the emergence of visual images.

I

INTRODUCTION

In recent years it has been proposed that a relation exists between the E.E.G. and imagery employed in thinking. It is claimed that while certain persons habitually employ visual imagery in thinking, others employ verbalization, the latter category being associated with irregular respiration accompanying sub-vocal speech. The "visualizers" are believed to have desynchronized, flat E.E.G. records with no detectable alpha rhythms and the "verbalizers" unusually persistent alpha rhythms (Golla *et al.*, 1943; Golla, 1948; Short, 1953; Walter, 1953; Mundy-Castle, 1951). These proposals have been criticized by Drever (1955) following studies of persons rendered blind early and late in life. In America, Walter and Yeager (1956) found it impossible to estimate the type of imagery used from the character of the electrical activity of the individual blind subject. Barratt (1956) found no evidence that suppression of the alpha rhythm provided a reliable index of the presence of visual imagery; imagery was found to be only one of many factors accompanying suppression.

At the present time it is widely suspected that desynchronization and disappearance of the alpha rhythm is a manifestation of the "level" of alertness, wakefulness, arousal, or attention of the organism, and that it is mediated through an activating mechanism in the brain-stem reticular formation. The evidence has been summarized by Magoun (1954) and by Jasper (1954). Low voltage fast records have been claimed to be particularly common in the presence of anxiety.

Golla, Short and Walter, however, claim that it is activity in those neurones subserving visual imagery that is responsible for desynchronization of the alpha rhythm. One worker who previously accepted the relation between imagery and the E.E.G. has, however, lately acknowledged that "the same effect can be caused by an independent factor, such as attention or the state of alertness" (Mundy-Castle, 1955).

The possibility that the association found between introspective reports of imagery and the alpha rhythm, could have an explanation other than that visual imagery causes desynchronization, does not appear to have received due consideration. It has long been realized that visual imagery emerges "at points where our thinking is baffled" (Betts, 1909), "when any slight check occurs, . . . in the absence of this the whole process is likely to run on to completion simply in terms of language" (Bartlett, 1932, p. 220). Humphrey (1951) has discussed the work of Ach and of Fisher, who found that as a mental task became more familiar and less of a problem, visual imagery decreased and verbalization increased.

Therefore it might be proposed that visual imagery may have been reported at the time of flattening of E.E.G. records, because both represented responses to difficulty, with a consequent rise in the "level" of attention, and that when language without visual imagery was reported, it was because there was but little difficulty. This view is consistent with Barratt's finding that where problem solving was accompanied by alpha suppression, there was a significant decrease in that suppression when the problem was thought over on a second occasion—when it might be presumed to be less difficult. The view that alpha suppression is associated with the level of wakefulness is consistent with the findings of the American workers, Walter and Yeager (1956), that successful recall of visual material was associated with low alpha amplitude during the session when the visual material was presented, the subjects being unaware at the time that they would be asked to recall it later.

Jasper (1954) describes how Einstein was found to show a fairly continuous alpha rhythm while carrying out rather intricate mathematical operations which, however, were fairly automatic for him. The alpha rhythm suddenly disappeared and he reported that he had just thought of a mistake in calculations he had made the day before.

II

Observations

An attempt has been made to follow the classification of E.E.G. records into those with no alpha rhythm, or "M" type, those with alpha rhythms that are normally responsive, the "R" type, and those in which the alpha rhythm is abnormally persistent, or "P" type, according to the definitions given by Short (1953). These definitions do not allow of any absolute differentiation between the groups. The common appearance of alpha rhythms just after hyper-ventilation, and briefly, on eye-closure, in those who otherwise lack alpha rhythms has been ignored.

(i) The records of some 200 cases where E.E.G. examination had been repeated after an interval were reviewed. Head injuries and other organic lesions were excluded and among the remainder were found four cases in which on one occasion a patient had had a record which fell clearly into the "M" type and on another occasion clearly in the "R" type. There were several others which, less definitely, could have been similarly classified.

(ii) E.E.G.'s were performed on individuals known to indulge spontaneously in certain particularly vivid varieties of visual imagery and the incidence of "M" types noted.

In 1880 Sir Francis Galton first described individuals who habitually experienced numbers in a particular visual pattern; these images he called "number-forms" and he published a collection of these (Galton, 1883). In the present investigation, out of 57 persons who were found to possess number-forms or analogous "forms" for months, the week, the alphabet or their families, it was possible to examine 33 electroencephalographically. It may be added that not only did these 33 persons experience these "forms," but at least 12 also experienced chromaesthesia and at least 11 could, with eyes closed, perceive so vivid a visual image, that on eye-opening, either a negative after-image or a projection of the original image was experienced with the quality of a real sensation. These latter individuals, and others, have been described elsewhere (Oswald, 1957). There is, therefore, no doubt that amongst the 33 should be found a considerable excess of "visualizers" with "M" type E.E.G.'s, if the existence of this relation were accepted.

In fact, of the 33, only 5 could be said to possess "M" type records. One had a "P" type and the remainder "R" type E.E.G.'s.

In addition a series of initial E.E.G's performed in this department on a consecutive series of 250 patients was examined and, on the same criteria, found to contain exactly 30 "M" type and 4 "P" type records, 4 which were unclassifiable (e.g. a record with generalized rhythms at about 20 cycles per second which blocked with eye-opening), while the remainder were "R" type records.

The incidence of "M" types was therefore practically the same in the group of "visualizers" as in the larger general population. It so chanced that the visual "forms" of the possessors of the five "M" type records were, if anything, poorer than the average in quality.

(iii) With 11 of the 33 "visualizers" described above, it was possible to carry out special E.E.G. examination while they performed various mental tasks involving imagery. In particular they were asked to "see" a certain number, month, etc., on their "forms." It was found that, with some tasks, the alpha rhythm would be momentarily blocked and then return. In other cases no detectable alpha blocking occurred. They had no difficulty in "seeing" these images for as long as required and reported their continuing presence long after any momentary blocking of the alpha rhythm. Thus, there was one subject who possessed a number, month, week, and alphabet-form, who experienced chromaesthesia, and after-images from images, and whose imagery conformed to the criteria of "eidetic" imagery. He was asked to see "101." His alpha rhythm, which was of a fairly continuous 50 microvolts, blocked for 5 seconds and then reappeared and continued without any noticeable difference from the normal for 30 seconds, during the whole of which time he claimed to see "101" in its position. Asked twice during this 30-second period if it was visible, he replied in the affirmative, without effect on the alpha rhythm.

The alpha rhythm of these subjects blocked when they *undertook some new or difficult task*, not while they viewed their visual images. This was further demonstrated in three cases by asking the subject to imagine he was travelling away from the building, along a route he knew well and to visualize the changing scene as he went. There was a momentary blocking of the alpha, after which it persisted steadily while he imaged the changing scene for about half a minute.

It was found that, if asked to perform arithmetical tasks, and to "see" the answer in its number-form position, simple sums did not block the alpha rhythm, though the visual image was present. More difficult sums would block it briefly, but when the answer was achieved and the visual image seen, the alpha returned. This was illustrated by one subject who possessed a number-form consisting of a series of half-loops

(a not uncommon variety). The majority of the possessors of number-forms deny involvement of their forms if asked to multiply 8 by 8, unless specifically asked to see the answer on their forms. This particular subject, however, was one who claimed that he automatically saw "64" in its position. While the record was being taken he was, in any case, asked to see the answer on his form and he affirmed that he did see it. His alpha rhythm, of 30 microvolts, was unaffected by the problem. A request to square 25, however, blocked his alpha rhythm for several seconds.

(iv) The claim that there exists a relation between regularity of respiration at rest, and the resting E.E.G. type, was examined. The principal evidence for such a relation is that of Short (1953).

The records of 100 patients on whom simultaneous E.E.G. and respiratory tracings had been made, were examined. The respiratory tracings were obtained by using a thermocouple near the nose. Some of our records done by this means are technically unsatisfactory and the 100 records were selected from a slightly larger group by an E.E.G. recordist who had no knowledge of the purpose of the investigation, nor why he was selecting 100 technically satisfactory records of respiration. The respiratory tracings were all recorded for one minute, five to ten minutes after the start of the E.E.G. recording. The thermocopule had been previously positioned and even if the patient guessed its purpose (which few do) he had had several minutes to settle down before a respiratory tracing was started, unknown to him.

It was ensured that when the judgement of the regularity or irregularity of respiration was made, there was no possible means of knowing the E.E.G. type and vice versa. A procedure of this type was not, apparently, followed by Short (1953).

Our 100 records were shuffled by assistants who numbered them 1 to 100. They were displayed one by one with only the respiratory tracings visible, the E.E.G. tracing being covered over. The respirations were judged by the writer to be regular or irregular. Another assistant, who alone knew the number of the record, wrote down the judgements. The whole procedure was repeated, again in ignorance of the E.E.G. and of the number of the record. The assistant then selected those records (18 in all) where two inconsistent judgements had been made, and again in ignorance of their numbers and E.E.G.'s, a final decision was made for each. The last 40 seconds of the one-minute's respiratory tracing, was examined in each case.

A similar procedure was then carried out with the E.E.G. tracings. Assistants exposed these one by one with only the E.E.G. visible. Records were judged to be "M," "R" or "P." A further category "X," was made for certain records of "R" type, which I believed might have been classed as "M" by, for instance, Golla, who has reported higher incidences of these records than we find. We almost never see "P" records. This is also the experience of the American workers, Walter and Yeager (1956). Our subjects sit in a chair facing the E.E.G. machine and two windows. If they were recumbent, in a plain darkened room, "P" types would no doubt be more common. Only eight of the E.E.G.'s needed a third and final decision as to their type (in seven cases this related to the "X" decision).

The decisions as to regularity of respiration and E.E.G. type had been recorded by an assistant, and not till all were finally made was his record sheet seen by me. The results are shown in Table I.

One E.E.G. which needed a third judgement of "type" had once been tentatively classed as "P." This happened also to be in a person with regular respiration.

TABLE I

RELATION OF E.E.G. "TYPE" TO REGULARITY OF RESPIRATION IN 100 PATIENTS

	"M"	*"X"*	*"R"*	*"P"*	*Totals*
Irregular ..	9	7	59	0	75
Regular ..	2	4	18	1	25

III

Conclusions

(1) The same individual may exhibit different types of E.E.G. record on different occasions. This would appear difficult to reconcile with a typology which would divide subjects into imaginal types on the basis of their E.E.G.'s.

(2) A group of persons known to possess vivid spontaneous visual imagery of a class which may be recognized with a fair degree of objective accuracy, did not contain an excess of flat E.E.G. records.

(3) The presence of a static or changing visual image was not accompanied by desynchronization of the alpha rhythm provided that the person concerned was not experiencing difficulty in achieving that image. Calculation, provided it was easy, did not suppress the alpha in cases where the end product of the calculation definitely involved visual imagery. These facts appear incompatible with the proposal of Golla (1948) that the presence of the alpha rhythm may "be taken as evidence of an arrested or non-cooperative state of the neurones subserving the mental process that is the basis of visual imagery."

(4) The claim that there exists a relation between the resting E.E.G. and resting respiration was not confirmed. There was no excess of regular breathers with "M" or "X" records. The only "P" type had regular respiration, as did the only other which had been tentatively classified as "P." Scrutiny of Short's figures showing α relation between the alpha type and respiration reveal that the relation is dependent on his "P" cases and that he found no significant excess of regular breathers in his "M" group. As previously stated, we see very few "P" records. Golla (1948) stated only that his "M" group showed "the predominantly regular type of respiration." It must be emphasized that all the above considerations of the alpha rhythm and respiration apply to records taken at rest. The writer fully accepts that subvocal speech is associated with irregular respiration. The results noted above suggest that persons with "M" type records may "talk to themselves" just as readily as those with "R" or "P" type records.

(5) Desynchronization of the E.E.G. during thinking occurs when difficulty arises. It is further recognized that difficulty of itself is the common precipitant of visual imagery in thinking. It is therefore proposed that suppression of the alpha rhythm may be explicable solely in terms of mechanisms controlling alertness, without reference to visual imagery. It is recognised that, of the human senses, vision is that which, when stimulated, most readily raises the level of attention.

In conclusion it is necessary to consider the theoretical premises of the belief that there exists an association between the E.E.G. and imagery. These are: (1) That thinking proceeds by the manipulation of images, as Short (1953) seems to believe. (2) That there exist imaginal types.

The first premise is one, the popularity of which has considerably decreased in the last 50 years and would be contested by many, e.g. Woodworth (1915). Humphrey (1951) has reviewed much of the experimental work on this problem.

The second premise is also one which has been doubted. Vernon (1937) wrote, "the evidence for clear-cut visual, auditory, kinaesthetic and verbal types is excessively small." Humphrey (1951) has referred to "the outmoded doctrine of imaginal types."

It is suggested that on both theoretical and experimental grounds, the claim that there exists a correlation between imagery and the E.E.G. merits scrutiny and re-evaluation.

I wish to record how greatly I am indebted to Group-Captain V. H. Tomkins, Principal Specialist in Neuropsychiatry, Royal Air Force, whose encouragement made these observations possible. I wish also to thank the several Electrophysiological Technicians of this department.

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NUMBER-FORMS AND KINDRED VISUAL IMAGES*

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A. INTRODUCTION

In 1880, Sir Francis Galton (4) first described persons who, when thinking of numbers, experienced a visual image, or "number-form," in which individual numbers occurred in constant spatial relationships to one another, and to the observer. The numbers might form a series arranged as some simple figure, or as some bizarre shape involving, perhaps, different planes and colours. Galton recorded his observations in a letter to *Nature* and, in consequence, received numerous further examples, so that he eventually published reproductions of 65 of them (5). He stated that 18 of these were derived from a group of 337 school-boys. He also described "forms" for the alphabet, months, days, and historical dates.

Calkins (3) summarised results gathered by her students concerning the incidence of "coloured thinking," and of mental "forms." In the first series of 525 persons, were 65 possessors of "forms," most of whom, it seems, had number-forms, apart from other varieties. In the second series of 203 persons, were 61 with "forms."

Pear (12), who reviewed the topic, reported that 35, of a series of 525 persons questioned, had "forms" of some kind. He did not state whether questioning was written or by verbal interrogation. Morton (9), using a questionnaire, found 20 possessors of number-forms in 867 school-children. McKellar (8), whose subjects appear to have given written answers to a questionnaire, found 14 possessors of "diagram-forms" of all kinds in 182 university students.

The writer has personally interrogated 300 young adults and questioned each about the possession of "forms." The subjects were personal acquaintances, nurses, and also patients referred for electroencephalography. Reports published elsewhere (10, 11) have dealt with certain manifestations of after-imagery, and also the electroenecephalograms, found in some of these subjects who had "forms." Certain other findings and conclusions may, however, be of interest.

* Received in the Editorial Office on December 23, 1958.

B. The Frequency of "Forms"

Among the reports quoted above, the second series of Calkin's stands out in that it reveals much the highest incidence of "forms." One infers that her students discussed the matter with the subjects, and that, evidently, increasing experience in framing questions led more "forms" to be detected. Similarly, the writer found only one number-form among the first 100 subjects questioned, but 5 in the second 100, and 11 in the third 100. The reason for this was that the writer was familiar at first only with Galton's descriptions and his statement of "the automatic appearance" of a number-form "whenever a numeral is thought of." This is, in fact, quite incorrect, and if one simply asks subjects to think of a few numbers, and then demands to know if they saw a visual pattern of numbers, then persons who do, in fact, possess number-forms, will reply in the negative. If they are of only average intelligence they will, moreover, fail to grasp that what one is seeking is something they experience under quite different conditions. What those conditions are depends on the individual.

In the present study, among 22 subjects found to have "forms" in the second 100 subjects questioned, and 32 in the third 100, there were, in all, 16 who possessed number-forms, 32 who possessed month-forms, 22 who possessed week-forms, 23 who possessed alphabet-forms, 11 with family-forms, and one with a time-form.

Among those last 200 subjects, were 31 women with 8 "forms" and 169 men with 46 "forms." There was no sign of the sex difference proposed by Galton.

C. The Conditions Under Which the "Forms" Are Experienced

The common circular type of month-form, with the months arranged around it, was most easily, and sometimes only, perceived, when a period of time was considered. Thus, "I have been living (or working) here since last October" would be accompanied by an awareness of the segment of the "form" corresponding to the period between the present time and last October. Similarly, a relative's birthday might be seen as "there." Merely to mention that August is a warm month was insufficient in some, while in others, the typical type of precipitant.

Another common, and often the only, circumstance under which a form appears, is when certain numbers, the family, or whatever it is, is thought of as a series. All those in the present study who possessed number-forms were asked to multiply 8 by 8, and subsequently all but four denied that they had seen "64" in its position on the form, though they could do so if

they tried. Not one of the subjects reported that they saw the "8s" on the form. If the numbers which constituted the problem were imaged at all, they occurred in the form in which a sum appears, on a blackboard, with lines beneath.

D. Are They Mnemonics?

Galton believed that number-forms arose in childhood as "mnemonics," as memory-aids which assisted the memorization of numbers.

It would seem more appropriate, however, to regard them as aids to the comprehension of abstract concepts, particularly the concept of sequence. The "form" provides a semi-concrete representation, allowing units with no inherent order to be arranged in orderly fashion. The representation may make use of familiar concrete objects, such as a clock-face, or, in the case of week-forms, it may resemble, for example, a tear-off calendar, somehow seen as if transparent and three-dimensional. Others, though three-dimensional, may be fantastic in shape.

In the present series, as in that of Calkins (3), "forms" to represent periods of time were commonest. Apart from a week-form, one man also possessed a time-form which was an endless spiral about six inches across, seen about six feet away. It was inclined slightly to the right of the vertical, the past being below and the future above. It appeared when the subject thought of, for instance, the concept of Eternity. The term "mnemonic" is obviously inappropriate.

Family-forms were not mentioned by those writers referred to above (3, 5, 8, 12). Several subjects, in the present study, associated their family-form with saying their prayers as children. One is shown in Figure 1.

This was seen as if it were a sort of solid histogram. It was possessed by a 38-year-old male mental nurse, by whom worldly, and especially intellectual, success was regarded as of supreme importance. Mother and father are on the left, then, highest of all in his regard, is his eldest brother, No. 3. on the "form," who helped the subject through examinations as a boy, and who is "a linguist and speaks 11 languages." Number 4 is a clerk who "hasn't bothered to better himself," No. 8, a sister "who has accomplished nothing," No. 9 is the subject himself, and No. 11, his youngest sister, a hydrocephalic mental defective. The subject knows that No. 3, the eldest brother, rose to his present height when the subject was aged 16 and was helped by this brother through school examinations. He agreed that Nos. 3 and 4 must once have been almost equal, and that, if, say, No. 6 now became an eminent public figure, then his place in the form would reach almost as high as that of No. 3. On the other hand, though the details of the form

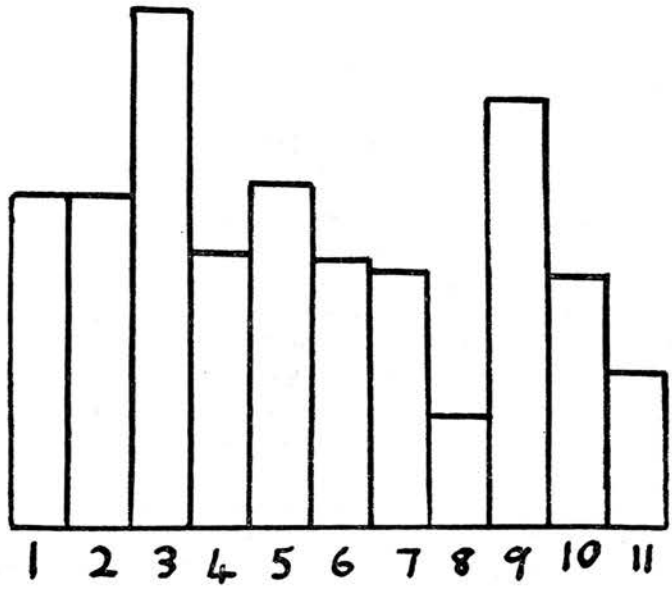

FIGURE 1
A FAMILY-FORM. MOTHER AND FATHER ON THE LEFT. NO. 9 IS THE SUBJECT. NO. 8 IS A SISTER "WHO HAS ACCOMPLISHED NOTHING."

are obviously not irrevocably fixed, yet he is positive that the basic plan, with the mother and father on the left, and a general downward slope to the right, could not alter. It is clear that this family-form was a concrete representation of abstract qualities rather than a memory-aid.

E. THEIR FLEXIBILITY

The family-form described had evidently a degree of flexibility. Another example was provided by an intelligent young woman who was certain that the summer months of the year, in her circular month-form, increased their share of the circle when, in her late schooldays, summer holidays became long and important.

Three week-forms were found, in which time was seen as a route which went high or low according to the "pleasantness" of the individual day, or the state of the week's finances—one man's was quite different in the Armed Services from its civilian precursor, because of the differences in the day of the week on which he was paid. Another took the form of, as it were, a path around a cylinder, about 15 yards across, the height on the cylinder varying with the day, and the time of the day. When high, life was bright-

green, when low, dark-green. Life was thus an endless circle for this subject, moving somehow inside a larger circle for the month, and a yet larger one for the year. The subject was an artist, with obsessional traits and a liability to periods of anxiety and depression when his usual routine of life was broken. When his whole way of life was changed, on entering the Armed Services, he re-evolved, after a period of brief psychiatric illness, a new way of life, and a new "up and down" circle. It is apparent that such a form as this was not a mnemonic.

F. Their Alleged Usefulness

In the years shortly before and after the beginning of this century, psychologists paid far more attention to studies of imagery than today. In many of the best circles it was accepted that images were the materials of thought, that thinking proceeded by their manipulation, and recall through the inspection by consciousness of images within itself.

The subjects of Calkin's series were college students, and her psychology students were very properly able to report that half of the subjects found their time-forms useful for placing and remembering dates, appointments, etc., and half of the possessors of number-forms thought they were useful for remembering dates and for mathematical operations. One wrote that she used her number-form in solving problems "because it helps me, but [let it be noted] my mind is closely occupied with the problem and is only dimly conscious of the form." Galton, apart from considering forms as mnemonics, did not consider other possible functions. One of his subjects, however, wrote "every number is always thought of by me in its own definite place in the series, where it has, if I may say so, a home and an individuality," but, "when I am multiplying together two large numbers, my mind is engrossed in the operation and the idea of locality in the series for the moment sinks out of prominence." That is to say, the actual task was carried through without the active manipulation of the visual image.

In the present survey, subjects were given simple tasks to perform and were then suddenly interrupted in that task and questioned about the presence of their "forms." Subjects with alphabet-forms were asked, for instance, "What letter comes three before P?" In the majority of cases the "form" did not appear and the answer was obtained verbally. In a few cases the letters were seen but, on close questioning, only one person claimed to have "read" them off backwards.

As previously stated, requests to multiply numbers sometimes, but not always, resulted in the appearance of the answer on a number-form, but the original numbers did not appear.

No subjects reported that any sort of manipulation of their number-form occurred in adding or multiplying. Indeed this is foreign to the nature of these "forms," which give fixed "homes" to numbers. Galton described a man who declared that he habitually worked out sums by the aid of an imaginary slide-rule "which he sets in the desired way and reads off mentally." No further details are given. Yet this man must, surely, as a child, have learned to do sums before he ever learned to use a slide-rule, so that his claim to calculate by visual-spatial means appears doubtful. One suspects that his imagery was merely a conscious parallel of those unconscious processes responsible for calculation, and not a precursor of them.

Morton (9), by mass questioning of schoolchildren, found 20 possessors of number-forms. The performance of the 20 children at arithmetic was significantly better than that of control children. There is, however, nothing in Morton's article to indicate that this was for any reason other than that only the more intelligent children were able to understand and give consistent answers to the series of questions put to them concerning their introspections, The figures, quoted earlier, suggest that he only detected a minority of those who possessed number-forms.

Morton assumed that the whole of the number-form must be visible at any one time. In the writer's experience, based on questioning, this is never the case, a *particular* number is seen with a sense of position. There may be a vague awareness of adjacent numbers only. Woodworth (14), in a discussion of imagery, emphasized that images of wholes are not seen with their details present, but only those parts of the whole, which are attended to, are seen in detail, and they are seen sequentially, as parts, but not altogether. A similar point was made by Klüver (7), discussing eidetic images.

Spalding and Zangwill (13) described a patient with a brain injury who subsequently exhibited defects of visual memory and imagery, presenting as (*a*) impairment of topographical sense, (*b*) a difficulty in copying and drawing from memory, and (*c*) impairment of the clarity of his number-form. In addition he suffered from dyscalculia, and the authors, who quoted Galton's statement that number-forms come into view automatically, whenever a numeral is thought of, concluded that the greater part of "the arithmetical disability is to be ascribed to impairment of the visual-spatial number-form." The examples of impaired arithmetic described, were incorrect addition of money, and the inability to multiply seven by seven.

As has been pointed out, number-forms do not come into view whenever a numeral is thought of, but only under special circumstances. Only five

of my subjects were asked directly if they could put three shillings and fourpence, or three shillings and a penny, on their number-forms. They were quite unable to do so, and indeed it would be unlikely that anyone could do so without turning the sum into pennies. As previously mentioned, the multiplication of eight by eight, did not involve number-forms, unless to give a position to the answer.

The writer therefore feels some reserve about accepting the hypothesis advanced by Spalding and Zangwill in regard to their case, and would prefer a point of view, such as that adopted by Brain (2) in regard to visual asymbolia, that though the latter may be frequently accompanied by other disorders, such as acalculia, visual agnosia, spatial disorientation, dysphasia, and hemianopia, these arise not because visual asymbolia and the other disorders are different manifestations of a single, more fundamental disorder, but because the anatomical basis of many functions is likely to be damaged by a lesion in the angular and supramarginal gyri.

G. Conclusions

It would appear that the term "mnemonic" is not really appropriate to mental "forms." Rather they appear to be aids to the initial comprehension of abstract concepts, giving to these a "sensory" character. Humphrey (6, p. 287) has pointed to the analogy between these "forms," and constructive acts of arranging the data of an abstract problem in some concrete, "sensory" way, expressed as a visual image.

These images appear only under special circumstances, and are static in nature. Their appearance is not inevitable; whether or not the image appears depends upon the particular task, as the Wurzburg experimenters, notably Watt, demonstrated (6, p. 70). They are images such as Aveling (1) found in his experimental study of the relations of images to meaningless or abstract concepts—images which acquire, by revival, a stable character, in turn giving stability to the concept they represent (1, p. 170). They are analogous in this respect to verbal names and verbal rote sequences. Like words, these images arise initially as constructive acts, acquiring stability with repetition. Unlike words, they serve no social function and are therefore less often revived.

The writer believes one may observe in the initial construction of these "sensory" data, the same type of "searching" for all available "sensory" cues, when some difficulty arises, that occurs with concrete tasks. In the latter case, percepts may be constructed out of information from the periphery, but with less concrete, more abstract, tasks, there is an increasing divorce

from peripherally supplied information, and the percept is constructed wholly of data from within.

H. Summary

Questions were put to 300 persons concerning their possession of those visual images known as "number-forms" or analogous images for the week, the year, history, or their families. At least one in four persons probably possess one or more of these images.

These visual images are liable to be experienced only if a series is under consideration, and not whenever the numeral, date, etc., is thought of. The term "mnemonic" is thought unsuitable and their alleged usefulness is questioned. They allow a "sensory," semi-concrete comprehension of abstract concepts.

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Reprinted from THE QUARTERLY JOURNAL OF EXPERIMENTAL PSYCHOLOGY,
Vol. XI, Part 1, February, 1959.

SHORTER ARTICLES AND NOTES

A CASE OF FLUCTUATION OF AWARENESS WITH THE PULSE

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The case is described of a man in whom certain percepts of central origin (after-images of hallucinations) fluctuated rhythmically with the arterial pulse. The phenomenon is explained in terms of contemporary theories of consciousness and the inhibitory effects of baroceptor impulses.

In a previous communication (Oswald, 1957) it was described how some persons may experience visual after-images not only after retinal stimulation but also after the prolonged contemplation of a voluntary vivid image (hallucination). One of those described, Subject Wi, is further discussed below. It is relevant to this case and to the interpretation suggested, to recall that Griffits and Gordon (1924) described the rhythmical increase and decrease of the size, intensity, or apparent distance of vivid images (hallucinations) with rhythmical fluctuations of vasomotor tone, of pulse rate and, by inference, of blood pressure.

Method and results

The general method used for the demonstration of the central after-images involved the subject concentrating on a sheet of blank paper and conjuring up a vivid image. When this appeared, the subject concentrated fixedly upon it for 30–45 seconds, and then looked away at a blank screen. On this screen an after-image of about the size expected by calculation at once, spontaneously and *effortlessly* appeared. In most subjects it would fade away within 5 seconds, but a few perceived such after-images longer. The subject with whom we are at present concerned, was exceptional in being able to perceive it far longer than anyone else (up to 30 or 45 seconds).

At the time of the initial observations, the writer's interest was wholly in the immediate size of these central after-images (which were measured) and little interest was taken in the fact that this one person alone remarked spontaneously that, after about 5 seconds, his after-image began to pulsate, going rapidly in and out and thus gradually shrinking smaller and smaller till it disappeared. This pulsation was not present in the primary image and was only faintly perceptible in the after-images of real visual patterns.

About a week after carrying out these measurements of after-images with the subject, it occurred to the writer to question him whether the pulsation was very rapid. When the subject indicated the approximate rate, the writer explained that he had wondered if it was much faster, if in fact it flickered at about 10 per second, the alpha rhythm rate. Lindsley (1952) has reviewed the evidence for fluctuation of consciousness at the rate of the alpha rhythm. The subject explained that it was much slower than that, remarking that, if anything, he supposed it would be about the rate of his heart.

Some five months later it was decided to investigate the pulsation of the central after-images further.

The subject was seated in a chair with electrodes on his head, a further electrode on the left arm (the casual remark being made to the effect that this was to measure changes in muscle tension in the arm), for recording the electrocardiogram. A further electrode was fixed to the wooden arm of the right side of the chair. It had previously been shown that tapping on the arm of the chair would, by this means, give a recordable signal.

The subject was in one room, while the electroencephalograph was in another. The rooms were separated by a corridor and two closed doors. The clicking of the electroencephalograph, each second, was inaudible in the room in which the subject sat.

He was told that it was intended to study the electroencephalographic and muscle tension changes occurring with his after-image. He was asked to try to tap in time with the inward phase of the pulsation. At the end of the session he was deliberately asked if

he had thought that his heart rate was being recorded. He replied that he had supposed the spring earth clip on his ear to serve for recording his pulse.

Five recordings were made in which he tapped in time with the ingoing phase of his after-image. Four of these, of 31 beats, 22 beats, 17 beats and 11 beats respectively, showed correspondence between his rate of tapping and his heart rate (*see* Figs. 1 and 2). The other showed correspondence for the first 10 of 30 beats.

FIGURE 1

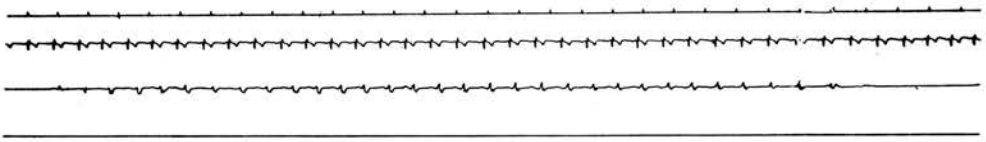
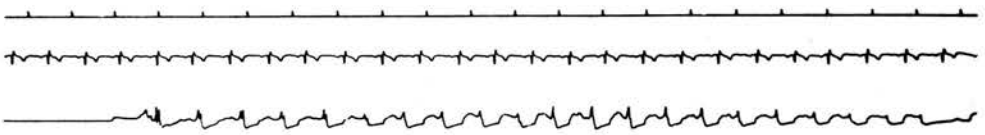


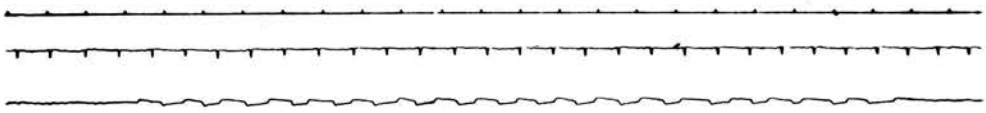
FIGURE 2



Showing, from above downwards, time-marker (seconds), electrocardiogram, and hand movements of subject in time with rhythmic decrease in size of his after-image

The subject was closely questioned in regard to whether he might be able to feel his heart beating, or feel or hear pulsations anywhere in his body, and, if so, whether he was aware of his after-images beating in time with them. He denied this. Further experiments were carried out by the writer and an assistant in which both tried to tap in time with their heart rates. None of these records showed any sort of correspondence between the rhythms, and indeed, it is a most difficult task. One may believe that one can feel faint beating sensations, but as soon as the arm starts tapping, attention becomes diverted to the much stronger sensations of the tapping, and any awareness of the heart or pulse is lost. Two similar recordings were made with the subject, who was asked to close his eyes and to try to tap in time with his pulse rate. Neither record showed any correspondence between the two rhythms (*see* Fig. 3). He affirmed that he had really tried, and concen-

FIGURE 3



Showing, from above downwards, time-marker (seconds) electrocardiogram, and hand movements as subject attempts to tap in time with his pulse. Note decreased decisiveness of movement

trated on feelings in his chest and eyeballs and he seemed to believe he had probably been tapping in time with these sensations, though agreed it was very difficult. Figure 3 indicates the decreased confidence of his tapping in the absense of a visual guide.

Discussion

It must be stated that there were no grounds for believing this man to be deliberately lying in saying that he was unaware of his pulse when sitting in the chair. He several times mentioned the after-image pulsation quite spontaneously, before the time when the writer showed interest in it.

Several hypotheses present themselves. One is believed to merit particular consideration—that the neuronal correlate of the after-image in the fore-brain persisted and that it was rhythmically "activated" by ascending influences from the reticular formation of

mid- and hind-brain. This accepts the contemporary belief that consciousness depends upon the interaction of, for instance, specific cortical effects of external stimulation and facilitative impulses ascending from the reticular formation.

It is known that with each pulse a sharp afferent volley of action potentials ascends from the baroceptors of the carotid sinuses (Bronk and Stella, 1932). These impulses reach the medullary and mesencephalic reticular formation and thence cause cortical inhibition (Bonvallet, Dell and Hiebel, 1954; Nakao, Ballin and Gellhorn, 1956). Increased carotid sinus pressure causes sleep-like states, not only in persons with hypersensitive carotid sinuses (Ferris, Capps and Weiss, 1935) with the production of electroencephalographic slow waves (Engel, Romano and McLin, 1944), but also in normal men (Schlager and Meier, 1947) and dogs (Koch, 1932). The effect can be independent of reflex bradycardia or hypotension.

Some support for the view advocated is offered by the following facts. Bonvallet, Dell and Hiebel (1954) described the rhythmic fluctuation of the level of cortical excitation with the rhythmic fluctuation of blood pressure (Mayer waves). These waves, often wrongly called Traube-Hering waves (*see* Schweitzer, 1945), occur in man. Intra-arterial blood pressure records in man have shown that in many persons there occur, as would be expected (Gunyton and Harris, 1951), oscillations of this homeostatic system about the mean, of the order of 20 mm. of mercury about every 10 seconds. In at least some circumstances these oscillations may synchronize with fluctuations of heart rate and skin vasomotor tone (e.g. Matthes, 1952). Roy and Sherrington (1890) suggested that this rhythm was responsible for "the rhythmic variations in the acuity of auditory and visual perception." Their suggestion was confirmed by several workers (e.g. Griffits and Gordon, 1924).

It is important to recall that a whole host of different internal and external influences summate to govern the level of activity of the ascending, activating reticular formation (and that the descending impulses arising during voluntary attention are probably of major importance). Such other factors can easily mask any effects of carotid-sinus impulses, as Bonvallet, Dell and Hiebel (1954) were at pains to emphasize. Therefore the spontaneous, *effortless* and wholly *central* after-images of the subject described would seem particularly open to the effects of carotid-sinus impulses. External stimuli can, of course, be responsible for both vasomotor changes and changes of cortical excitation, as Ackner and Pampiglione (1957) have described, but as these authors pointed out, so can internal stimuli and among these, impulses from the carotid sinuses merit consideration.

The observations described were carried out while the writer was serving in the Electroencephalography Department, Princess Mary's Royal Air Force Hospital, Halton, Buckinghamshire and during the period of tenure of a British Medical Association Research Scholarship. It is a pleasure to acknowledge the encouragement of Group Captain V. H. Tompkins, Principal Specialist in Neuropsychiatry, Royal Air Force.

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Reprinted from "Electroencephalography and Clinical Neurophysiology Journal"
Vol. 11, No. 2, May 1959.

CLINICAL AND LABORATORY NOTES

Edited by R. S. SCHWAB, M.B. and H. FISCHGOLD, M.D.

A PROPOSED ORIGIN OF THE NON-SPECIFIC EEG RESPONSE

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(Received for publication: June 25, 1958)

INTRODUCTION

It is the purpose of the present communication to draw attention to an EEG artifact that seems hitherto undescribed or at least unrecognised. Electroencephalographers are familiar with the potentials produced by rotational sliding of the eyeball in its socket. In comparable manner potentials are produced by rotational sliding movements between the scalp and the skull. These potentials are similar to the so-called "non-specific EEG response", "spike at the vertex" or "waking K-complex" (Davis 1939; Gastaut 1953; Bancaud *et al.* 1953; Roth *et al.* 1956; Larsson 1956) and it will be pointed out that potentials of scalp movement must inevitably accompany the "non-specific EEG response" on many occasions, if indeed the phenomena are not one and the same.

MATERIALS AND METHODS

The observations of principal interest were made on four volunteers capable of voluntary ear and scalp twitches. The results with all four were identical and similar to results produced with several other subjects whose scalps were moved manually.

In these four individuals recordings were carried out, using, on different occasions, saline pad electrodes and stick-on electrodes. As the results were the same with each method, the potentials could not be attributed to rocking movements of the pad electrodes. The stick-on electrodes used were Ediswan silver cup electrodes, filled with electrode jelly and fixed with collodion in the normal manner to scalp which had been degreased with alcohol. On different occasions a six-channel and an eight-channel Ediswan electroencephalograph were used. Recordings were made with a pen gain of 7 mm. for a 50 μV. signal, with time constants of 0.3 sec.

Bipolar recordings were made both with eyes open and eyes closed and with various combinations of electrode positions. Except for a slight shifting of the point of phase reversal from time to time in the same subject, results by all the methods used were entirely consistent.

RESULTS

It was found that when a subject gave a quick backward twitch of the ears (the scalp always moves simultaneously), a potential which varied in size according to the size of the twitch was produced. It was maximal in the midline, with a phase reversal at the vertex (see fig. 1). With leads linked to amplifier input connections in the conventional manner (Hill and Parr 1950) the main deflection was downwards from the front electrodes and upwards from the rear electrodes. However, quite often the main deflection was preceded and followed by deflections in the opposite direction, the former being small and rapid, the latter larger and slower. Very small ear twitches caused large EEG deflections. Indeed, owing to the coarseness of voluntary control of this movement effected by "vestigial" muscles, it was difficult to make twitches with sufficient delicacy to produce a vertex potential of less than 50 μV.

DISCUSSION

These potentials are very similar to the "non-specific EEG response". Bancaud *et al.* (1953) considered the question of whether the EEG responses they observed could be artifacts due to ocular movement, or electromyographic potentials, but justifiably rejected such possibilities. The artifacts described in this paper cannot be so readily dismissed, for not only are they similar in appearance, but would be expected to occur simultaneously.

Gastaut (1953) pointed out that the "spike at the vertex" was a component of the startle reflex (Landis and Hunt 1939). Among the components of this reflex is a very brief, widespread contraction of superficial muscles about the head and neck, the blink component of which is most obvious. Larsson (1956) showed that "latencies, stimulation thresholds and frequency-sensitivities for both the non-specific EEG response and for the blink component

1 The work described in this paper was done during, initially, a British Medical Association Research Scholarship, and later a Beit Memorial Fellowship for Medical Research.

in the muscular startle reaction are, if not equal, at any rate of the same order'' and both could occur as conditioned responses.

Landis and Hunt (1939) studied the startle reflex pattern with the aid of high-speed cinematography. They pointed out that a backward twitch of the ears and scalp was a constant feature of the mammalian pattern and were able to detect it in the human. They did not state its latency but it might be expected to be similar to that of movements caused by other superficial muscles (upwards of 20 msec. for the eye blink and 50 msec. for mouth widening).

There are, therefore, grounds for believing that the backward twitch of the ears and scalp is the cause of the ''non-specific EEG response'', which, it may be pointed out, has never been clearly demonstrated electrocorticographically. Detailed further resemblances between the two responses may be enumerated.

1. As shown in figure 1 the ear twitch produces EEG deflections similar in form and distribution to the non-specific EEG response, even including the small initial positive component emphasized by Roth *et al.* (1956).

2. The voltages are comparable, of the order of 50-150 μV.

3. The durations are comparable. Bancaud *et al.* (1953) give the duration of the EEG response as 80-400 msec. The deflections produced by voluntary ear twitches are of the order of 200-400 msec. for the main component. It is to be expected that the reflex twitch detected by Landis and Hunt with their high-speed cinematography would be more rapid.

4. Surprise and increased intensity of stimulus (of any sense modality) enhance both the EEG response (Gastaut 1953) and the muscular startle pattern (Landis and Hunt 1939).

5. The EEG response is enhanced on the spastic side in hemiplegia (Gastaut 1953) i.e. over the normal side of the brain. While this is hardly capable of satisfactory explanation if it is believed to have a cerebral origin, it could have been predicted if thought to be caused by a twitch, for the muscular startle pattern is enhanced on the spastic side (Landis and Hunt 1939).

It may be proposed that the onus of proof must lie upon those who wish to continue to believe in the cerebral origin of the ''non-specific EEG response''.

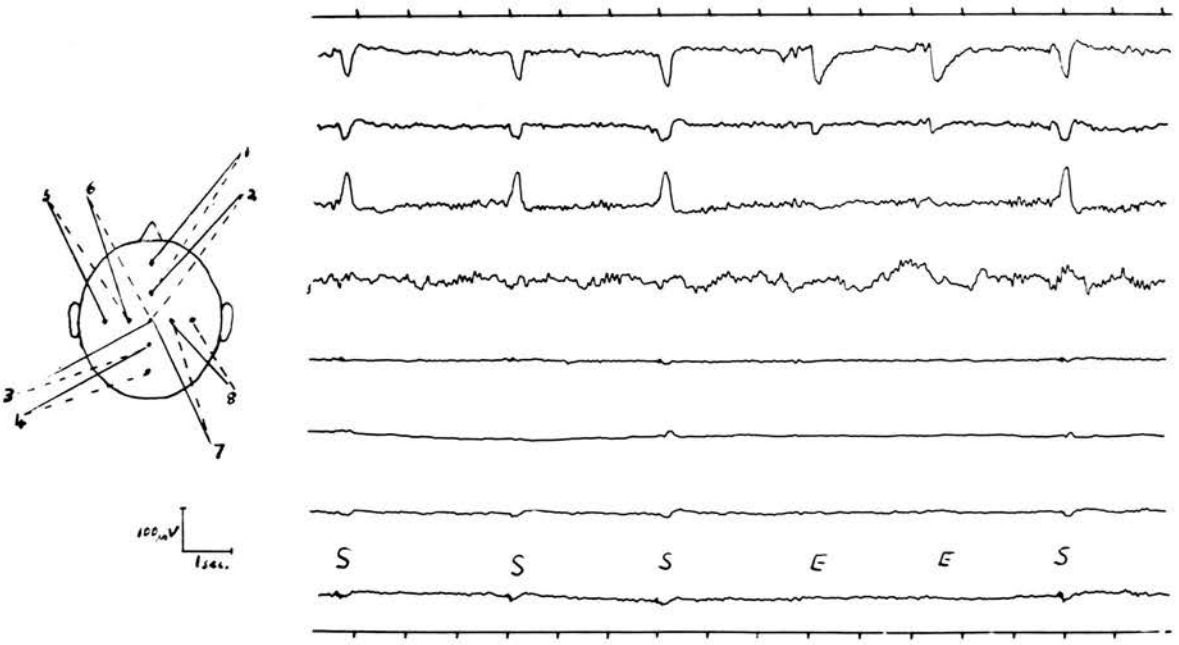

Fig. 1

Silver cup electrodes filled with electrode jelly and fixed to the scalp with collodion were used. The record was taken with the eyes closed.

S = scalp movement
E = eye blink

Note that the scalp movements cause artifacts with a phase reversal in the mid-line at the vertex. Note that the main deflection may be preceded and followed by lesser ones in the opposite direction.

Even could such proof be obtained it would still be necessary to show that many such responses were not distorted by the artifacts of scalp movement which would, at least sometimes, be expected to occur simultaneously.

It should be borne in mind that Landis and Hunt (1939) adduced evidence for the mid-brain origin of the startle pattern, a source which has also been assumed for "non-specific EEG responses" (Larsson 1956).

Scalp movement can also produce, if slow, artifacts like the K-complexes of sleep. There is pathological evidence that the latter are of intracerebral origin (Grossman 1949). It has also been claimed that they appear in subcortical recordings (Sem-Jacobsen *et al.* 1953). In one of the writer's patients, sleeping K-complexes were increased over a large, unilateral, skull defect, the edges of which were slightly adherent to the scalp.

Roth *et al.* (1956) reported that in some stages of light sleep, neither the waking nor the sleeping K-complexes could be elicited. This could be interpreted as indicating a different origin of the two forms of K-complexes. Furthermore, the main deflection in the K-complex of sleep has a more anterior voltage peak than is the case with the "waking K-complex" (Roth *et al.* 1956). Finally, in contrast to the waking responses, the sleeping K-complexes have been reported as decreased on the side opposite to the hemiplegic side of the body (Grossman 1949). While there is no reason to question the intracerebral origin of the K-complex of sleep, unless those of the waking state can be studied in cases where the scalp is absent or immobilised, it may be suggested that no deduction can be drawn from them about cerebral function.

SUMMARY

Evidence is brought forward to indicate that the "non-specific" EEG response may be an artifact caused by the rotational sliding of the scalp on the skull which occurs as part of the startle reflex.

I wish to thank Dr. V. H. Tompkins, Dr. Denis Williams, Professor R. C. Oldfield and Dr. P. Glees for their encouragement and for facilities.

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Reprinted from the BRITISH MEDICAL JOURNAL
June 28, 1958, *vol. i, pp.* 1515–1516

6

DEPRIVATION OF PARENTS DURING CHILDHOOD

ITS FREQUENCY IN SOME CONTEMPORARY YOUNG SERVICE MEN

BY

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The statistics presented below are offered not only because of differences which are apparent between the diagnostic groups but in the belief that they may serve as a background against which to assess the stress to be laid on a history of separation from a parent.

Bowlby (1951) reviewed the evidence pointing to the very frequent history of separation of the child from the parents in those with behaviour disorders. He regretted the paucity of controlled series in the case of psychiatric patients. An American study by Oltman *et al.* (1952) would appear to meet this objection, though a comparable controlled series from this country does not seem to have been reported.

Among the statistics reported below, some relate to patients with a history of head injury. A number of workers have claimed that accident-prone persons tend to come from broken homes (e.g., Csillag and Hedri, 1949; Tillmann and Hobbs, 1949). Biesheuvel and White (1949) in a study of accidents among South African Air Force pilots recorded that "there was a highly significant difference between the accident and control groups in respect of the numbers of cases where the father had died."

Method

The statistics were collected from Royal Air Force personnel who attended for electroencephalographic examination. The medical officers by whom the patients were referred were wholly unaware of the inquiry and therefore it cannot be argued that they tended to send especially those with a history of deprivation. Only patients aged

18–29 were questioned. The questions were read out from a standard written form and the answers recorded in code and on punched cards. The standard form of questioning was rigidly adhered to in order that it might be possible to refute any complaints that improper questions had been asked. Though details of divorces and brutality were often volunteered, they were never asked for.

The questions were, "Are both your parents alive?" If the answer was in the negative the age of the patient at the time of the death was ascertained. "Can you remember, to the best of your ability, whether at any time during your youth or childhood you were separated from one or other of your parents for a period of about six months or longer, for any reason at all—for instance, your father in the Army, you evacuated, you or your parents in hospital, or *any other reason whatever?*" Where there was a history of separation, only the first episode of six months was recorded. Thus, multiple separations over the years, or continued separation, were arbitrarily classified under the age when the first separation occurred.

In most instances these questions were put, and the information was recorded, by the electrophysiological technicians of the department. They were carefully instructed in what to do, and were reliable and intelligent—four were university-educated, two were awaiting entrance to a university, and the other three were of comparable ability. Before the statistics were recorded the patient's diagnosis was noted from the request form for an E.E.G. The diagnoses were arbitrarily grouped as E for epilepsy, P for psychiatric case, O for "other," HS for head injury (self-responsible), and H for head injury (not responsible).

It is important to note that the E group differs from those encountered in civilian hospitals. Nearly all had had only one or two fits, and the first of these had generally occurred in the Service. Chronic epileptics, whose families might have been disturbed thereby, were not encountered. A few neurotic patients who received the benefit of any doubt in the diagnosis of "black-outs" are probably included (which merely makes the observed differences from the P group more real).

The P group contained especially patients classifiable as of "inadequate personality," referred according to the varying tastes of their medical officers—especially if headaches, "dizzy spells," and aggressive behaviour were prominent.

The O cases were assorted neurological patients and cases of definite migraine, cardiovascular syncope, etc. It was expected that this group might contain a fair sprinkling of neurotics, and for this reason the hypothesis at the outset was that there would be a significant difference between the E and P groups and that the O group should be ignored.

The groups H and HS were made up of (*a*) those referred on account of head injury, and (*b*) those referred primarily for some other reason, but who, on routine pre-electroencephalography questioning, stated that within the previous two years they had had a head injury of sufficient severity to cause unconsciousness. The head injuries were also classified into groups—"driver," "passenger," "sport," and "other." These figures are not presented owing to the smallness of these groups. The decision on whether the individual was responsible or not for his injury was always made before the family history was taken; and while the P, E, or O diagnoses were occasionally altered on the basis of later information, the H or HS diagnoses were never altered. Emphasis was placed on severity in allotting responsibility, and, while passengers were mostly H cases, only two drivers were treated thus leniently.

Hypotheses at Outset

The initial hypotheses were: (1) that there would be an excess of cases of deprivation in the P group when compared with the E group; (2) that there would be an excess of cases of deprivation in the HS group when compared with the H group. As previously stated, the O group was to be ignored. The groups E and H were both expected to be normal control groups, and, as there are no cross-diagnoses between these two groups, they could be added if desired.

The distribution of the different varieties of deprivation in each diagnostic group is shown in the Table.

Frequency of Deprivation of Parents

Diagnostic Group:	P	E	O	HS	H
Total No. of cases in group	271	119	245	245	91
No history of deprivation	114	67	132	118	49
Deprivation of any kind at any age	157	52	113	127	42
,, ,, ,, ,, ,, ages 0–5 inclusive	87	32	61	68	23
Deprived by death or separation for 6 months from *both* parents at ages 0–5 inclusive	36	5	18	16	2
Mother dead	20	11	11	20	8
Age at mother's death, 0–5 years	7	3	6	10	3
,, ,, ,, ,, 6–10 ,,	4	4	3	3	0
,, ,, ,, ,, 11–15 ,,	3	4	0	2	2
,, ,, ,, ,, 16–20 ,,	6	0	2	5	3
Separated for 6 months from mother	62	9	34	31	17
Age when separated, 0–5 years	33	4	18	12	8
,, ,, ,, 6–10 ,,	19	5	10	12	5
,, ,, ,, 11–15 ,,	10	0	6	7	4
Father dead	42	17	32	36	10
Age at father's death, 0–5 years	17	7	12	11	5
,, ,, ,, ,, 6–10 ,,	6	2	4	6	0
,, ,, ,, ,, 11–15 ,,	10	2	11	9	3
,, ,, ,, ,, 16–20 ,,	9	6	5	10	2
Separated for 6 months from father	110	34	81	87	27
Age when separated, 0–5 years	63	21	41	50	16
,, ,, ,, 6–10 ,,	33	11	30	29	7
,, ,, ,, 11–15 ,,	14	2	10	8	4

Discussion

No elaborate analysis of the figures is justified when the initial hypotheses are borne in mind. However, it may be noted that the first of these hypotheses is confirmed. If the P and E groups (there were no cross-diagnoses between these groups) are compared in respect of those with a history of deprivation of any kind and those without such a history, there is a significant difference between them ($\chi^2 = 6.7$, $P = 0.01$). This difference could be interpreted as indicating that deprivation of parents increases liability to mental ill-health (though no figures for a probably more important history of unhappiness, brutality, etc., are available). Alternatively, it could be claimed that the mentally unstable tend to exaggerate a history of parental loss, or that they inherit their predisposition from parents whose own instability contributed to the breakdown of home life. Or, again, it is possible that the mentally healthy forget or gloss over parental deprivation. The difference between the two head injury groups is in the expected direction, but is small and not significant.

The effects of wartime evacuation of children, and war service by the father, on these statistics, which were collected during 1956 and 1957, are very obvious in the high incidence of separation from parents in the early age groups.

The Table shows also a high recorded incidence of death of fathers, and, to a less degree, mothers, for the age group 0–5 years. In this connexion it should be remembered that this group covers six years of life, unlike the other groups. Wartime casualties, particularly among the fathers, must also be considered. Even so, the incidence of maternal deaths in this period still seems high, and may be accounted for (*a*) because in the case of the P and HS groups such maternal loss does, in fact, cause behaviour disturbances, or (*b*) because of unreliability inherent in the nature of the inquiry. While it is reasonable to compare the different groups, the absolute figures may need to be treated with caution because of the rigid form of the questions asked. There was no mandate for questions about illegitimacy or adoption ; and, indeed, to ask such questions of a patient referred for E.E.G. examination because he had crashed his aircraft and sustained a head injury might have led to justifiable complaints. Consequently the figures for the 0–5 age group, in particular, may be distorted owing to untrue or evasive answers.

Summary

The frequency of deprivation of parents during the childhood of a series of young Service men was investigated in relation to diagnosis. Significantly more of those who were psychiatric cases had a history of depri-

vation than cases referred on account of isolated epileptic attacks.

There was not a significant difference between a group of persons responsible for accidents and a control group.

The investigation was possible because of the co-operation of the electrophysiological technicians of the department. I thank Group Captain V. H. Tompkins, principal specialist in neuropsychiatry, Royal Air Force, for his encouragement. The present investigation was among those carried out during the period of tenure of a British Medical Association Research Scholarship.

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GAINSBOROUGH PRESS, ST. ALBANS.

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DELIBERATE RE-HYPNOTIZATION AFTER THE PATIENT'S REFUSAL

BY

IAN OSWALD

Reprinted from

THE JOURNAL OF MENTAL SCIENCE

Vol. 105, No. 440, July 1959

DELIBERATE RE-HYPNOTIZATION AFTER THE PATIENT'S REFUSAL

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A CASE closely comparable with that reported below does not appear to have been described, possibly because of the ethical problems involved. Watkins (1947, 1951), in the description of two cases, has made the most nearly related contribution to the old debate as to whether an unwilling subject can be hypnotized. His cases were both trained subjects who were offered money rewards to attempt to resist the formal induction of hypnosis before an audience. They were both successfully hypnotized. The fear-inspired unwillingness of the patient to be described would appear to have been in a rather different category.

CASE HISTORY

The patient was a 21-year-old female typist of average intelligence. She had suffered from habitual nocturnal enuresis till the age of eight and occasionally thereafter. Her chief complaint, however, was of a liability to incontinence of urine by day under conditions of excitement or under emotional tension of any kind—an extreme form of a common symptom. Her admission for psychiatric treatment was precipitated by an unfortunate example of this incontinence. She was playing table-tennis in mixed company, and during a brief break in play was lightly springing about on her toes. One of the men present asked, jestingly, "What's the matter with you? Can't you hold your water?" Such was her embarrassment that she was immediately incontinent of urine, a fact which was obvious to those present.

It was thought that she might benefit from a technique for giving increased control of urgency, using hypnosis. With this object she was interviewed by the writer, and, after a discussion of "emotional tension", it was proposed that she could be taught to relax and, by this means, control her disability. The emphasis was on relaxation and no mention of hypnotism was made.

Suggestions of relaxation were made and gradually a fairly deep trance achieved with the successful production of rigidity of limbs, analgesia of the hand and deafness. The demonstration of the control of sensation was used to explain to her how "relaxation" could help to control sensations and would be used to control bladder sensation. In order to facilitate subsequent re-hypnosis the suggestion was made that, "Whenever you sit in a chair and look at me and I clap my hands, you will immediately return to the state in which you are now". Amnesia for this suggestion was then induced to increase its likelihood of success (Erickson and Erickson, 1941). The trance was then terminated and the patient returned, after further brief discussion, to the ward.

In the course of the evening of that day the patient fell into conversation with another patient. The latter had lately been hypnotized by another psychiatrist and mentioned this fact, whereupon the other realized that she too had been hypnotized. Furthermore, the amnesia which had been intended to cover one specific suggestion, in fact covered almost the whole of the session. The patient, having only the lay person's knowledge of hypnotism, was greatly alarmed and distressed to find that she was unable to recall what had happened between the time of the initial conversation with the writer and the end of the interview. She confided her fears to the ward sister.

The next morning the patient refused to have any further treatment. She was interviewed by the writer in the presence of the ward sister and the nature of hypnotism simply explained in an attempt to reassure her. She remained quite unmoved in her attitude however, despite assurances that it was impossible to make a hypnotized person do anything if she was not willing (this is interesting in view of the subsequent happenings). She did not respond to a request to ask any questions she liked. She showed marked hostility, and when asked why, replied, "I'm frightened of you."

A considerable practical and ethical problem was obviously present. If her refusal

of hypnosis was respected, her amnesia would remain. If however, despite the ethical objections to such a course, a successful attempt to re-hypnotize her were made, her amnesia could be removed, her fears and distress relieved, and the treatment originally planned could be continued. The decision taken, rightly or wrongly, was that to leave her with a patch of amnesia associated with a fear of something unknown could have so adverse an effect upon her as to justify an attempt at re-hypnotization.

The patient was therefore interviewed later that day by another psychiatrist with the ward sister present, and a few moments after this began, the writer entered the room on the pretext of discussing an electroencephalogram. On the way out of the room the writer casually asked the patient, "Can you still not remember?" As she replied she looked at the writer, who was carrying his hands lightly together before him and who then very quietly and unobtrusively clapped his hands. The patient suddenly diverted her gaze, and continued to reply that she couldn't remember. Then she hesitated and became glassy-eyed. She said, "My mind's going all queer." A second hand-clap then accompanied the instruction, "Close your eyes and try and remember", after which a third hand-clap was made. The patient had by now entered an obvious trance.

"You can remember now."

"My arm is all stiff." Pause. "My hand" (she pinched it as in the first session).

Further explanations and suggestions were now made. "You are not frightened now are you?" "No." Asked if she would continue the treatment she assented readily. Suggestions were then made to the effect that she would have no more fear, and would indeed look forward to the treatment sessions with happiness because her disability was to be relieved. The trance was then ended, after which she appeared confused as if some further spontaneous amnesia was present.

When seen the next day she was happy, smiling and co-operative, and remained so whenever seen in the seven months which followed, except that four weeks after the original episode she stated that she still felt apprehensive before being seen. Hypnotic suggestions rectified this.

Treatment

She was discharged from hospital, as she could easily be seen as an out-patient. She was seen weekly for the next six weeks and every three or four weeks thereafter.

Post-hypnotic suggestions were used to bring about analgesia of any part of the body when she said aloud, "No pain in my (hand, etc.)." Then when she just "thought" the words and finally when she "wanted" it without actually saying any words to herself. Similarly she was taught to render the analgesic part normal again at will. This removal and return of sensation was repeated many scores of times, and practised by the patient during each day till full voluntary control of the perception of discomfort in the superficial tissues was attained. After three weeks of this, post-hypnotic suggestions were given to the effect that later that day, whenever she felt the need to pass urine, she would at once voluntarily abolish the sensation causing that need, so that she would only go to the toilet at tea-time and bed-time. She was instructed not to attempt this except on that one day.

When seen the next week she reported success and further hypnotic suggestions were given—that she would be able voluntarily to abolish her sensations of urgency, and so control the desire to micturate, on all days, and at all times, in the future.

She was seen at intervals for another five months, and throughout this period reported that she now regularly micturated only three times per day, instead of a dozen times or more. Her voluntary control of pain perception also continued. She expressed herself delighted. She became engaged, got married, and left the district. An attempt to contact her later was not successful.

Comment

The patient was a particularly good hypnotic subject. The present case would lend support to the view that if such a person can once be hypnotized, providing appropriate suggestions have been made during that initial trance,

hen subsequent re-hypnosis can be achieved despite strongly motivated refusal ›y the subject. This would also probably be true with an unscrupulous ıypnotist.

The eventual successful treatment of this patient justified, it can be argued, he decision deliberately to re-hypnotize her after her refusal. It is very doubtful f a successful courtship, culminating in marriage, would have been compatible vith the patient's previous disability.

The achievement of voluntary control of perception of discomfort, in the vriter's experience, is only occasionally as stable as in this case. It is entirely compatible with the view that to perceive is as much an active, constructive esponse as is a motor act which is subject to voluntary control.

The case is similar in some respects to that of a patient described by ones (1956), whose urinary frequency and incontinence were successfully reated by a conditioning procedure. The method adopted in the present case, nowever, avoided the disadvantages attendant upon repeated catheterization.

SUMMARY

A case is described which contributes to the debate whether an unwilling subject can e hypnotized. The patient was a female who suffered from embarrassing urgency and ncontinence of urine with excitement or emotional tension. She was hypnotized by xplanations about "relaxation". Suggestions were given for re-entering a trance at a and clap.

Amnesia for the trance appeared. Discovery, later, that she had been hypnotized aused great fear and despite all reassurances she refused further treatment. The ethical roblem being fully considered, she was re-hypnotized, despite her refusal, by a hand clap. uggestions dispelled her fear.

Hypnotic suggestions were used to teach voluntary control of perception of discom- ort, including bladder sensations. Her disability was completely relieved during a seven nonth follow-up during which she pursued a successful courtship, culminating in marriage, fter which contact with her was lost.

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P A R T II

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Discriminative Responses to Stimulation During Human Sleep

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[*Reprinted from* BRAIN, *Vol. 83, Part III, 1960, pp. 440–453*]

MACMILLAN AND CO., LIMITED
ST. MARTIN'S STREET, LONDON

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ST. MARTIN'S PRESS, INC.
103 PARK AVENUE, NEW YORK 17

1960

DISCRIMINATIVE RESPONSES TO STIMULATION DURING HUMAN SLEEP

BY

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INTRODUCTION

IN recent years a great deal of evidence has indicated that ascending impulses, from the brain-stem reticular formation, govern the electrical signs of wakefulness or sleep that can be recorded from the cerebral cortex, and that collateral afferents from all sensory paths play an important role in exciting the reticular formation. Bremer (1954) proposed that during natural sleep the level of activity of the reticular formation could be raised by cortico-fugal impulses. In support of this, both Bremer (1954) and Brain (1958), have quoted the alleged superiority of an auditory stimulus of personal significance (such as a person's name or the cry of a child) compared with an auditory stimulus lacking special importance for the sleeper. How certain, however, is this alleged superiority? It could, for instance, be thought that any spoken name might momentarily awaken the sleeper, who then, from secondary cues, or by "scrutinizing" some immediate memory mechanism, decides that his own name has been called and so makes an overt response. The present report deals with studies designed to examine these problems.

SENSORY DISCRIMINATION DURING SLEEP

The German physiologist, Burdach (1830), wrote, "The psyche isolates itself during sleep . . . nevertheless . . . we are not always awakened by the mere sensory force of the impression, but by the psychic relation of the same; an indifferent word does not arouse the sleeper, but if called by name he awakens. . . . The mother awakens to the faintest sound from her child . . . hence the psyche differentiates sensations during sleep . . .

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we may be awakened by the lack of a sensory stimulus if it relates to the presentation of an important thing . . . the miller wakes when the mill stops." These statements won the approval of both Freud (1913) and Pavlov (1955).

Adrian (1937) said, "A loud noise which has no importance for the sleeper affects the auditory nerve as usual but produces only a transient and localized response in the cortex. The cry of a child . . . will give a message of much smaller intensity yet will set the whole brain in action." "The unfamiliar noise wakes the sleeper because the afferent message reaches the cortex and is there judged as important. The cortex signals back to the diencephalon and the rapid spread of activity ensues." Now the cry of a child is "new," or "novel," compared with the rattling of a door, the noise of the wind or the singing of the birds. Habituation, or disappearance of response on repeated stimulation is a property of stimulus-response mechanisms of all degrees of complexity (Humphrey, 1933; Oldfield, 1937). The arousal response can follow sensory stimulation during sleep. If that stimulation is repeated (as in the case of the rattling door) the arousal response would be expected to disappear even if the only sensory paths involved were collateral afferents to the reticular formation. Characteristic of habituation is the phenomenon of dishabituation—reappearance of response when a degree of novelty is introduced into the stimulus. Hence a new noise (the cry of a child) would be expected to cause arousal, even if the only sensory paths involved were collateral afferents to the reticular formation, and (contrary to Adrian's quoted remark about the "unfamiliar" noise) cortical discrimination need not be predicted as essential. Experimental confirmation of this has been provided by the work of Sharpless and Jasper (1956) who found that in cats, habituated during natural sleep to one tone, dishabituation to a new tone still occurred in cats with extensive destruction of auditory cortex, just as in normal cats. Dishabituation to a *pattern* of tones was apparently impaired, however.

Rowland (1957) has reported experiments in which cats were used, and which showed that if, for instance, a cat, in a series of waking training trials, received an electric shock after each presentation of a 25 clicks per second stimulus, then habituation of the EEG arousal reaction during sleep to that stimulus alone, was much slower than to a 240 c/sec. tone stimulus. Rowland also stated, though without reporting details, that finer discriminations, e.g. between 384 c/sec. and 480 c/sec. could be achieved.

One of the most striking features of the human sleep EEG, is the presence of K-complexes following sensory stimulation (Davis *et al.*, 1939; Roth *et al.*, 1956).

In an experiment with a human volunteer (Oswald, 1958), a conditioned emotional arousal response to the middle one of three tones, of 530, 690

and 790 c/sec., was established. When the three tones were later presented during sleep, K-complexes to the "important" tone occurred with a much greater frequency than to the other tones, the difference being statistically significant.

Discrimination Between Names

Method

In order to eliminate the factor of novelty, subjects were given the task of responding selectively to a particular name among a steady series of names spoken while they were asleep, the degree of novelty of that particular name being no greater than that of neighbouring names.

A list was drawn up of 56 male and female Christian names and, included among these, were the names of all the students and research staff of the Institute of Experimental Psychology. A tape recording was then made, names being spoken urgently and with slightly unnatural clarity of individual syllables, in a randomised order and such that the intervals between names were 5, 7, 4, 8 and 6 seconds, repeatedly. Each name occurred singly eight times in all and then, without any break, the names were twice recorded doubly (Alan, Alan! Stephen, Stephen!. . .). There were 448 single and 112 double name stimuli.

When the recording was made, we did not know who would volunteer to be subjects. We could, therefore, hardly have spoken those subjects' names specially loudly—a possibility which is, in any case, largely provided for by the method of using control names, described below.

Each subject remained awake for the whole of the night prior to the day of the experiment, in order to ensure sleepiness. At about 7 p.m. we met, and, in some cases the subject at once received orally a 4 grain tablet of sodium hexobarbitone. Silver cup electrodes, containing electrode jelly, were then fixed to the scalp, to the palm of the left hand, and to the dorsum of the left forearm, as shown in fig. 1. The three

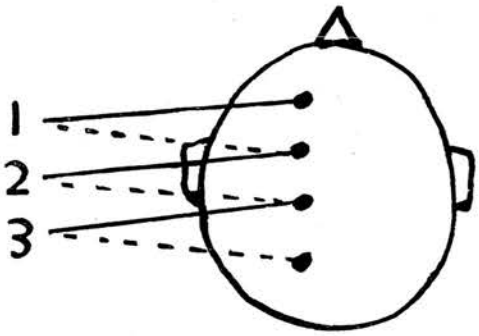

Fig. 1.—The electrode positions used in all experiments.

electrode pairs on the scalp were sufficient for the picking up of K-complexes, and the pair on the limb for recording hand movement, galvanic

skin responses (GSRs) and, usually, the electrocardiogram. The respiration was recorded in four cases, using a mercury-in-rubber tube, but this proved unsatisfactory owing to subjects turning over in sleep.

The subject was told that he was to go to sleep and that, during sleep, whenever his own name was spoken, or that of one other, control name, he was to awaken and clench his left fist. It is important to note that subjects were used as controls for each other, the "other," or control name to which each was to respond being the "own" name of one of the other subjects, the arrangement being that the "other" name, in each case, had no particular significance for the individual.

The subject then lay down, covered by a blanket, on a mattress in a small, dimly-lit room, next to the room in which the electroencephalograph was housed. About 2 feet from the subject's head was a loudspeaker which led from a Brenell Mark V tape recorder. A microphone was placed in front of the loudspeaker and connected to the electroencephalograph as a stimulus indicator (this did not give a reliable indication of the relative loudness of different stimuli, which were, in fact, fairly uniform). The EEG paper speed was generally 1·5 cm./sec., though occasionally 3 cm./sec. On the electroencephalograph available, all recordings unfortunately had to be done with a 0·03 sec. time constant.

When the subject had settled down, the tape recorder was started, the names being played at the intensity of loud conversation. The subject, in this way, grew accustomed to the constant succession of names before he fell asleep, and had opportunities to practise his responses. Some subjects fell asleep within a few minutes, others not for several hours, and three (excluded from consideration) not at all. If, after a couple of hours, the subject was still not asleep, a further 4 or 8 grains of hexobarbitone was administered orally. When the tape ended, it was rewound and played again and again in such a way that subjects slept for a major proportion of one to three playings of the tape. Each name, as it was played, was written in pencil at the bottom of the recording paper.

Assessment of Results

The EEG record of each experimental session was taken and arranged by one of us (A. T.) on a table which had been specially prepared so that the record could unfold from the right and be slid along to the left, some four feet of record being exposed, except for its bottom couple of inches, which were always covered over by a long shield. Consequently, any observer, though able to see, on the top channel, the stimulus signals (from the microphone), *could not know which names were represented*, since these were covered, and the stimulus signals themselves, as may be seen in the various figures, gave no indication of which name was involved.

The record was slid from right to left, one of us (I. O.) marking in pencil on the record the letter "N," "K" or "R" below each stimulus

signal, according to whether, in his opinion, the particular stimulus had provoked, respectively, no EEG response, a K-complex, or some apparent lesser response undeserving of the title K-complex.

In order to merit the judgment "K," there had to be an obvious, large, slow wave and anterior 12 c/sec. waves. A mere sharp wave was not accepted as a K-complex and was called "R." The judgments "N," "R" or "K" were only made when the EEG record showed the individual to have been in sleep of suitable depth. Light sleep, characterized by low voltage 4–6 c/sec. waves, in which rather poor K-complexes could sometimes be seen, was ruthlessly excluded. Very deep sleep, characterized by high voltage 1 c/sec. waves was not scored for EEG responses either, since K-complexes cannot be seen during this. Scoring of EEG responses was thus confined to the C and D stages of sleep as defined by Loomis *et al.* (1937).

Each record was later worked through and, for each example of the person's own name, and also the "other" name, there was noted the judgment "N," "R" or "K" which had been previously written on the record, together with the judgment which had been made for the immediately preceding name.

In addition, the channel from the arm and hand was scrutinized for muscle spikes and movement artifact. Owing to the infrequency of these during sleep, it was necessary, for statistical purposes, to note, in addition to movements occurring after each crucial name, the number of movements during the period of the preceding *ten* names. The scoring of how many movements there had been, could not be done in ignorance of the names involved. Such scores are reasonably objective and any small bursts of muscle spikes, or movements, were counted.

Results

The combined results for ten subjects, one of whom had two sessions, are given below. Some excerpts which are shown are to be regarded only as illustrating the findings. Owing to occasional spontaneous awakenings, statistical treatment of results is essential.

Hand movements.—Hand movements which occurred after 131 examples of "own" name during sleep, at least as deep as the C stage of Loomis *et al.* (1937), numbered 33, and while these were predominantly large, co-ordinated responses associated with awakening (e.g. figs. 2 and 3, Plate LXX), some represented vague stirrings during sleep (e.g. figs. 4 and 5).

$$\text{Hand movements to "own" name} = \frac{33}{131} \quad \text{.....(a)}$$

Hand movements that occurred in the period of the preceding *ten* names numbered only 17, and these were mostly short bursts of small muscle spikes related to readjustments of limb position (which occur in

sleep without awakening) or other small artifacts (e.g. after "Hugh, Hugh" in fig. 5). Since there were two instances in which the sleeper awakened, in the preceding ten names, other than to one of the crucial names, and had remained awake at the time of "own" name, these are counted in equation (b) below, though they have to be excluded from equation (a) above.

Hand movements to the ten names preceding "own" name

$$= \frac{17}{1330} \quad \text{(b)}$$

Similarly, in the case of hand movements to the "other" name (e.g. figs. 6 and 10).

Hand movements to "other" name $= \frac{15}{124}$ (c)

Hand movements to the ten names preceding "other" name $= \frac{21}{1270}$ (d)

The differences between (a) and (b) and also between (c) and (d) are, statistically, very highly significant. In addition "own" name is significantly more often followed by movement than "other" name, for if one examines the difference between (a) and (c), $\chi^2 = 7{\cdot}1$, $n = 1$, $P < {\cdot}01$.

K-complexes.—These results are of particular importance since they throw light on the possible argument that the non-specific, noise qualities of any name might act, via collateral afferents to the reticular formation, to cause an arousal response, which might be manifest as a K-complex, and that *subsequent* to this, there might take place complex analysis of some immediate memory (the storage devices of which might be independent of the level of wakefulness). In fact, the results indicate that discriminative analysis *precedes* arousal as manifested by the K-complex.

The total number of possible K-complexes under consideration is less than the total of possible movements, since, as previously mentioned, very deep sleep must be excluded when K-complexes are considered.

K-complexes to "own" name $= \frac{73}{110}$ (e)

K-complexes to immediately preceding name $= \frac{37}{110}$ (f)

The difference between these is highly significant, $\chi^2 = 13{\cdot}4$, $n = 1$, $p < {\cdot}001$

K-complexes to "other" name $= \frac{48}{109}$ (g)

K-complexes to immediately preceding name $= \frac{38}{109}$ (h)

The difference between (g) and (h) is not significant. "Own" name was more likely to provoke a K-complex than "other" name, the difference between (e) and (g) being statistically significant, $\chi^2 = 10{\cdot}1$, $n = 1$, $P < {\cdot}01$.

K-complexes in the absence of movement.—As would be expected, when a movement followed "own" name, there was nearly always a preceding K-complex, in fact, in 30 of the 33 instances. The other 3 cases had been scored as "R," though they could, no doubt, have been labelled "K" by a different observer, their slow wave components being small and their fast components more striking. However it was not the case that the high proportion of K-complexes to "own" name was due solely to those instances where movement also occurred, for, if the latter are excluded, we find that "own" name was still significantly more likely than the preceding name to evoke a K-complex. Furthermore, in these cases, there were not available to the scorer the occasional cues as to the name, visible in the EEG signs of full awakening that at times followed movement to "own" name.

K-complexes to "own" name when no movement $= \frac{43}{77}$ ..... (i)

K-complexes to immediately preceding name $= \frac{25}{77}$ ..... (j)

The difference between (i) and (j) is significant ($\chi^2 = 8{\cdot}5$, $n = 1$, $P < {\cdot}01$).

The onset of names spoken twice at a time.—As already described, the final part of the tape recording was made up of names spoken twice at a time. These followed the others without any break or any noticeable change of loudness. In the figures the convention of adding the mark ! after the name has been adopted to indicate that it was spoken twice. On 11 of 26 occasions this change in the pattern of repetitive stimulation caused awakening or brought about lighter sleep.

Response to Names Played Forwards and Backwards

Aim

We set out to discover whether, in a person who had been asked to "listen" to names during his sleep, such names, when recorded on tape, would be more disturbing if the tape was played in its normal direction, than if played in reverse, for the latter destroys the familiar phoneme structure and the significance of speech.

Method

The Brenell tape recorder, previously mentioned, is an instrument which can play or record upon the upper and lower tracks of magnetic tape simultaneously but quite independently.

The 56 names previously mentioned, were again spoken, to give a recorded total of 112 names, except that, on this occasion, the interval

between each name was 15 secs. The top track of the tape was used. The spools were then lifted off the tape deck and reversed, so that the track with the recorded names on it was at the bottom, and what was now the top track was clear. The tape machine was now started so that the names on the bottom track were played back in reverse at 15 second intervals. Seven to eight seconds after each of these, each name was again spoken, as before, being recorded on the top track. When this was finished we therefore had a tape on which were 224 names, half of which were recorded in one direction, alternately with the other half recorded in the opposite direction.

The tape was subsequently played in one direction to sleeping subjects, both tracks being played at as nearly the same loudness as possible. After the tape had been run in one direction, it was removed and reversed. The amplifiers for each track were now also changed over, so that if the list of names previously played forwards on the top track had had, for instance, the benefit of louder amplification, then that same list of names would have the same loudness while played backwards on the bottom track and vice versa. The tape was now played again. The output leads from both amplifiers were joined, in parallel, to the same loudspeaker.

In order to be able to compare "names forwards" with "names backwards," it was, of course, necessary that the subject should be asleep for nearly the whole time while the tape was played in both directions and that sleep should be of fairly uniform depth. Three subjects used had to be rejected because they were awake for the greater part of the second run of the tape, but seven others satisfied the requirements.

Assessment of Results

The EEG responses to the name stimuli were scored as before, except that, in addition to "N," "R" and "K," an additional category of "K+" was used to denote the more majestic, polyphasic K-complexes, the principal criterion borne in mind being the presence of three or more large slow waves in the K-complex (an example is shown in fig. 7).

TABLE I

	Forwards				*Backwards*			
Subject	N	R	K	(K+)	N	R	K	(K+)
Joan	61	30	119	(54)	132	31	47	(12)
Peter ..	128	30	36	(3)	141	24	33	(0)
Neville ..	36	24	126	(24)	85	31	69	(8)
Ian	54	16	114	(36)	99	26	57	(12)
Alex	76	49	69	(8)	104	33	60	(6)
Barrie ..	110	24	64	(2)	132	20	50	(3)
Geoffrey ..	28	24	146	(32)	56	27	114	(13)
Totals ..	493	197	674	(159)	749	192	430	(54)

In the table the scores of "K" responses include polyphasic K-complexes, the numbers of which are indicated in brackets under (K+).

Results

K-complexes.—The scores of EEG responses are shown in Table I. The small differences, with some subjects, between the numbers of backwards names and forwards names, were determined by the moments of falling asleep and waking up, for some subjects would awaken for a few seconds, or longer, several times during the playing of the tape, and scoring of EEG responses was not, of course, done for these periods.

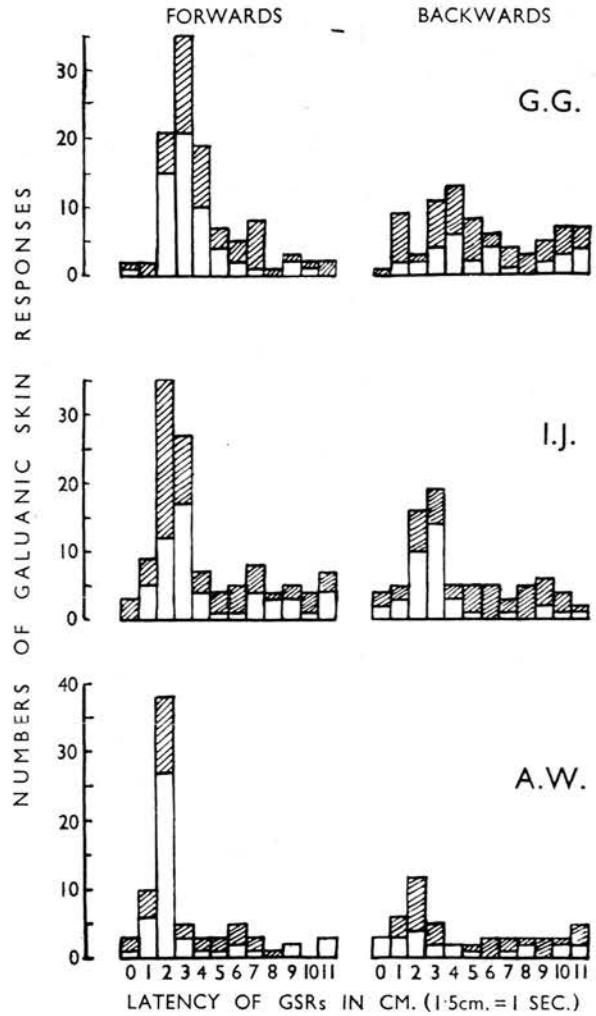

FIG. 8.—Three subjects showed occasional galvanic skin responses, during sleep, to the forwards and backwards names. The latency of these has been plotted in terms of centimetres of recording paper. The unhatched areas relate to the playing of the tape in one direction, the hatched areas to the playing in the other direction. It may be noted that forwards names evoked far more GSRs of 2–5 cm. latency than did backwards names.

It will be seen that names played forwards to these subjects, during sleep, were far more provocative of K-complexes than the identical stimuli played backwards, the difference being very highly significant. In addition, given that a K-complex occurred at all, it was significantly more likely to be a polyphasic one if the stimulus was a name played forwards rather than backwards. Only 54 of 430 were polyphasic, in the case of backwards names, whereas 159 of 674 were polyphasic in the case of forwards names ($\chi^2 = 20{\cdot}5$, $n = 1$, $P < {\cdot}001$).

Galvanic skin responses.—3 of the 7 subjects, at present under consideration, produced GSRs to the name stimuli. In fig. 8 are shown the distribution of GSR latency to backwards and forwards names. It will be seen that names played forwards are far more evocative of GSRs during sleep than are the same names played backwards, the difference lying in those responses having a latency period of about 1–3 secs. between the beginning of the stimulus and the beginning of the response. One of the 3 subjects produced GSRs most readily during deep sleep and, during this, obviously responded differentially to names forwards and backwards (fig. 9).

Control Studies

There appeared to be two possible factors which might have influenced the results, namely the effect of slight differences in preceding silent intervals, and the effect of the altered physical pattern of stimuli. In fact, the average silent intervals preceding the forwards and backwards names, respectively, were about the same, and a check of the results of the earlier experiments, where different intervals had been deliberately used, showed this factor to be unimportant.

A name like "Gordon" tends to be spoken in such a manner that it is louder and more abrupt at its onset than at its termination. In the extreme case a sound of very gradual onset would be relatively ineffective as a stimulus. We therefore decided to investigate this by using meaningless stimuli having such a loudness distribution.

The stimuli used consisted of alternating patterns of increasing and decreasing loudness of a 400 c/sec. tone. Each pattern had three components of equal duration, each separated by 15 decibel steps. Each component followed the preceding one instantaneously. Patterns were at seven second intervals and of 0·5 sec. total duration. Results with 2,400 stimuli, from six subjects, showed that the meaningless patterns of *increasing* loudness, far from being the less effective were, in fact, slightly but significantly the more effective in causing EEG arousal signs during sleep. Further, the frequency distributions of GSR latencies, in 4 subjects who produced these to the tone patterns, were in sharp contrast to fig. 8, there being but a small difference between increasing and decreasing patterns, with increasing patterns of loudness being slightly the more

evocative. These results with *meaningless* patterns are probably related to the fact that, when heard during wakefulness, the increasing loudness patterns appear, subjectively, greater total stimuli, being experienced as a pattern, while the decreasing loudness patterns cause an illusion in which only the first component is experienced—a manifestation of "short term auditory fatigue" (Licklider, 1951).

General Discussion

Sensory Discrimination During Sleep

It has been clearly demonstrated that sleeping subjects can make complex discriminations between repetitive auditory stimuli. They can, for instance, discriminate between meaningful words, and the identical auditory stimuli made meaningless by the reversal of direction of play of a tape recording. These latter complex stimuli apparently have little arousal effect compared with familiar and meaningful speech sounds. Language functions are commonly held to necessitate a functioning cerebral cortex.

We may suggest that, during sleep, signals from sense organs continue to reach the cerebral cortex and that the latter continues to receive, in some degree, ascending facilitation from the brain-stem: that complex analysis of the significance of the sensory signals can be made, and that, if the stimuli are personally significant, cortico-fugal signals to the brain-stem may evoke, in turn, arousal signs which may be electroencephalographic, autonomic or behavioural. Our subjects have evinced less evidence of discriminative ability in deep sleep, when the cortex presumably receives least ascending facilitation, than in sleep of medium depth. Nevertheless, some degree of that ability persists even in deep sleep (figs. 9 and 10).

Given that the discriminative analysis of the repetitive, sensory material involves cortical neurones—and the experiments of Sharpless and Jasper (1956) and Diamond and Neff (1957) support the belief that the reticular formation alone could not do this—then our suggestion of cortico-fugal impulses to the reticular formation is consistent with the beliefs of Adrian (1937) and the work of Bremer and Terzuolo (1953) and of Segundo *et al.* (1955).

We have, of course, confirmed the lay belief that a person can set himself the task of awakening to some particular stimulus and can, in fact, succeed in that task to some extent. Similarly, some people will experience more sleep disturbance than others from a given type of stimulus if they attach special significance to such stimuli, for instance, the man who is worried about his nocturnal jerks may be more easily awakened by them and so remember more of them (Oswald, 1959).

Habituation During Sleep

Contrary to Roth *et al.* (1956), habituation of the K-complex response to recurrent auditory stimuli during sleep has been observed, being much more marked in some subjects than others.

Consideration of, for instance, fig. 3, will emphasize that the absence of some electrical response of the brain to repetitive stimulation does not imply that the principal mechanism normally underlying habituation in the mammal is a more or less peripheral blockade of sensory inflow, mediated by centrifugal fibres, as Hernández-Peón *et al.* (1957) appear to propose. Rather, failure of response may be present *after cortical analysis* of the stimulus has already established its lack of importance for the individual and vice versa. Another instance of this seemingly steady scrutiny of sensory signals, to which no obvious attention is devoted, has been provided by our colleague, Moray (1959), again using personal names.

The EEG K-complex

In Section III it has been shown that the K-complex is not only a response which appears subsequent to some analysis which has established the significance of the auditory stimulus for the individual, but that, in addition, the more significant the stimulus, the larger the K-complex. It is strange that an especially potent stimulus should evoke the most big, slow waves—which are normally the concomitant of the deepest sleep.

SUMMARY

The work arose from the proposal that when a sensory stimulus causes arousal from sleep, cortical analysis of the personal significance of the stimulus precedes that arousal.

In order to test the belief that a person can arouse especially to his own name, it is essential to eliminate the factor of novelty of type of stimulation. Therefore a tape recording was made of 56 names, spoken urgently, at intervals of 5, 7, 4, 8 and 6 seconds, each name occurring many times in all, with also, a series of names spoken twice at a time. Sleep-deprived volunteers fell asleep while the tape was being played, having been instructed to clench their fist in response to their own and a control name.

Some subjects showed a remarkable capacity to wake up from sleep to their own name, the latter being significantly more likely to provoke a K-complex than other names, even if the subject failed to respond overtly. Ability to arouse to some other pre-selected name, though manifested, was less marked. Statistical analyses have been made of EEG responses, on the basis of ratings of the latter made in ignorance of each particular stimulus. The responsiveness largely, but not entirely, disappeared in very deep sleep characterized by high voltage 1 c/sec. waves.

In a further series of controlled experiments it was found that personal names recorded on tape were far more likely to evoke K-complexes if played in the normal, forwards direction to a sleeping person, than were the identical stimuli played backwards. Furthermore, if a K-complex did occur, it was significantly more often a polyphasic one if the evoking name was played forwards rather than backwards. Names played forwards also evoked significantly more galvanic skin responses during sleep.

These observations are thought to indicate that prior analysis of the significance of a sensory stimulus occurs during sleep, which, owing to the complexity of the required discriminations, must presumably involve the cerebral cortex, and that subsequent arousal depends on cortico-fugal excitatory signals to the reticular formation.

ACKNOWLEDGMENTS

It is a pleasure to acknowledge the encouragement and facilities given us by Professor R. C. Oldfield, Dr. P. Glees and Professor E. G. T. Liddell. We wish also to thank many of our friends for practical help, especially Mr. T. Oates, Mr. N. Moray, Dr. R. Davis and Miss H. Ross and, of course, all our volunteer subjects.

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PLATE LXX

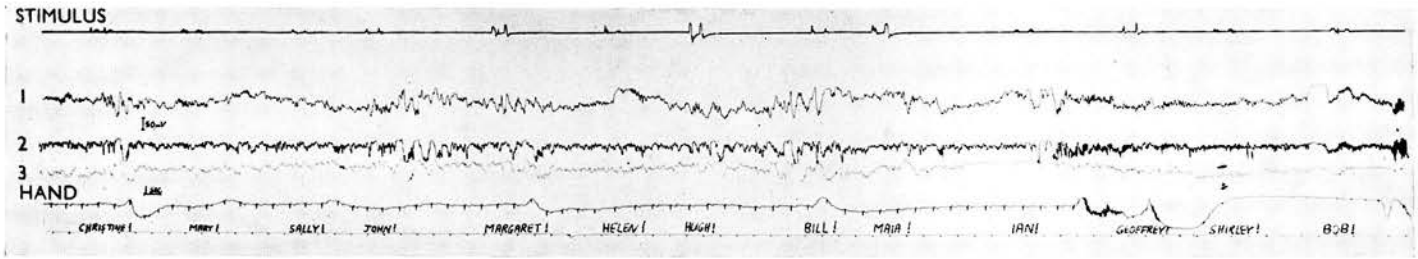

FIG. 2.

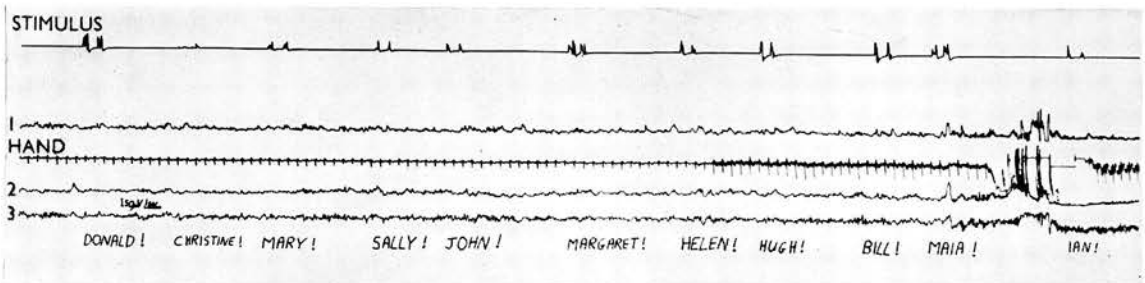

FIG. 3.

To illustrate article by Ian Oswald, Anne M. Taylor and Michel Treisman.

PLATE LXXI

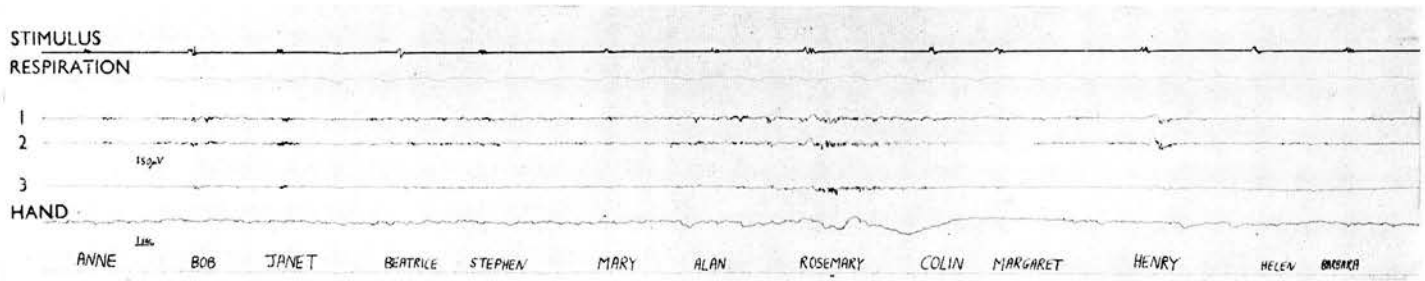

FIG. 4.

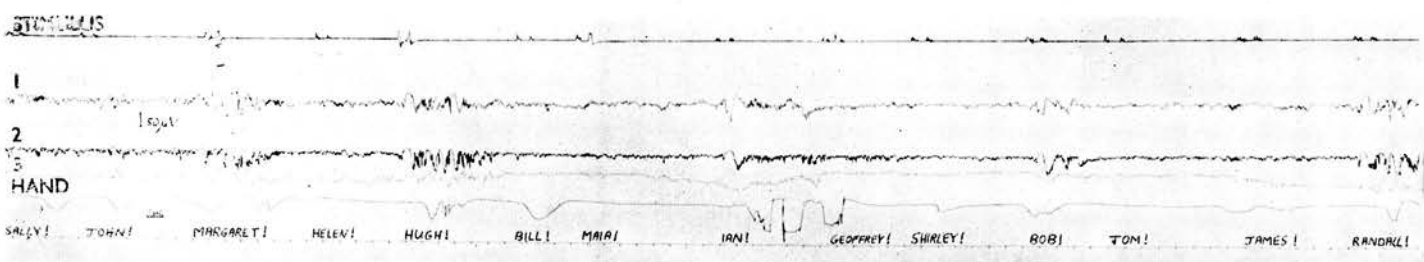

FIG. 5.

To illustrate article by Ian Oswald, Anne M. Taylor and Michel Treisman.

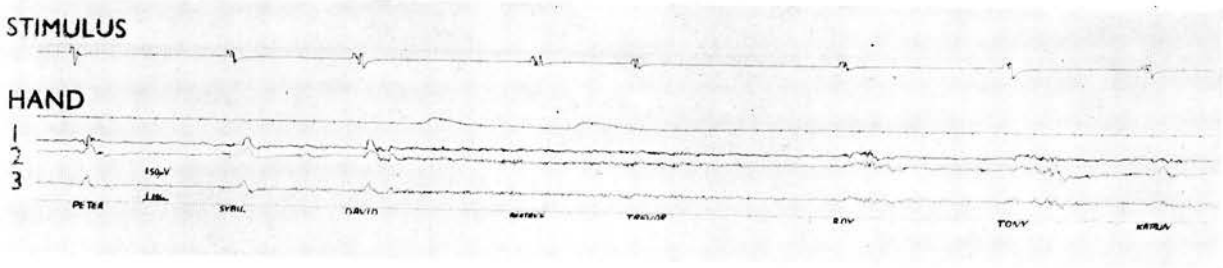

FIG. 6.

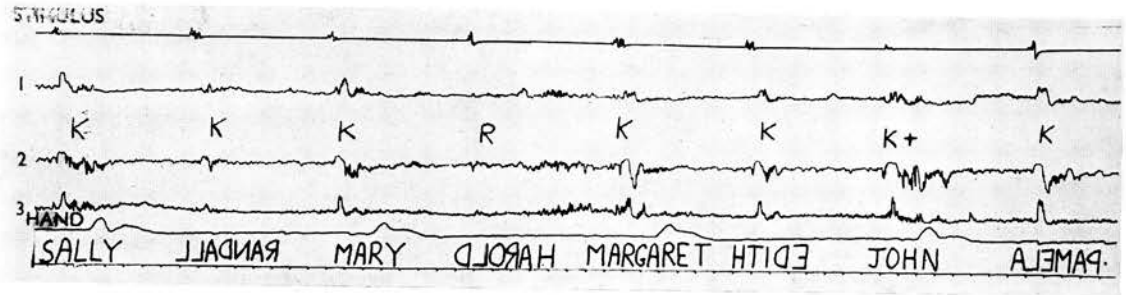

FIG. 7.

To illustrate article by Ian Oswald, Anne M. Taylor and Michel Treisman.

PLATE LXXIII

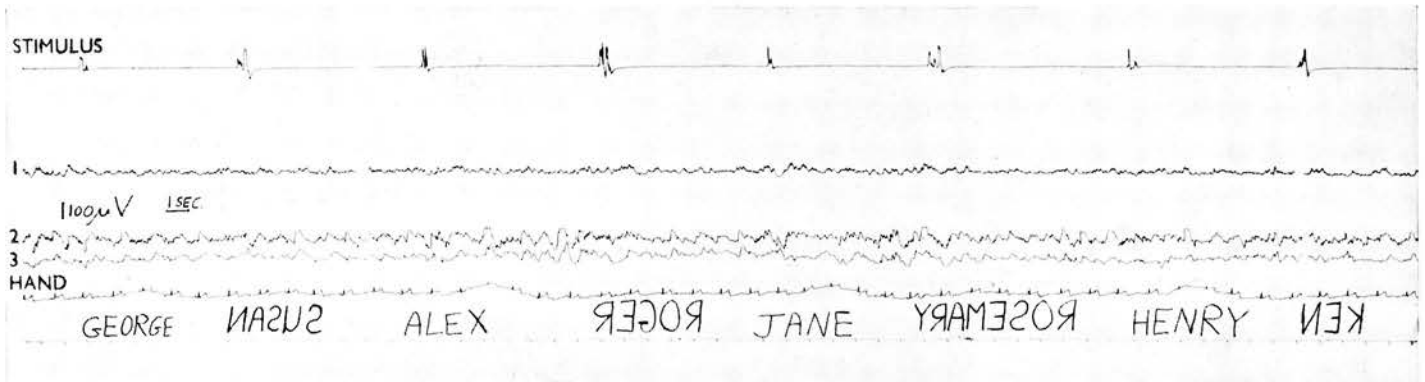

FIG. 9.

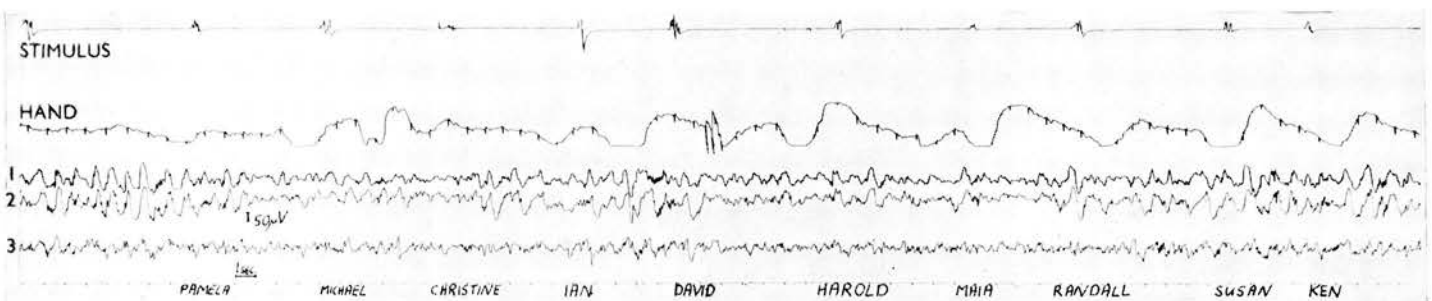

FIG. 10.

To illustrate article by Ian Oswald, Anne M. Taylor and Michel Treisman.

LEGENDS FOR PLATES

PLATE LXX

FIG. 2.—This subject's name was Ian. In the section shown the names were each being spoken twice. Though quite deeply asleep (Loomis D stage) with runs of high voltage slow waves independently of stimulation as well as K-complexes, he awakens to his own name.

FIG. 3.—This girl's name was Maia. The segment of the tape recording is the same as that in Fig. 2, but she awakes to her own name. Her record was, throughout, remarkable for the small number of K-complexes. In this section she was sleeping, with random 1–6 c/sec. waves generally and anterior spindles of faster activity, until, half a second after the first, "Maia," a large slow wave, maximal near the vertex, was seen, followed by generalized 12 c/sec. waves, then a galvanic skin response, then hand movement, then return of alpha rhythm. The amplification of the hand channel had happened to be turned up shortly before, but this was a silent event and not relevant to the awakening.

PLATE LXXI

FIG. 4.—This girl's name was Rosemary. She is in medium depth sleep, with frequent anterior spindles. She is one who showed marked habituation of K-complexes, which occur only occasionally. Her own name provokes a K-complex which stands out, and the channel from the hand, which has a base-line wobble, shows a sudden big wobble.

FIG. 5.—The same subject as Fig. 2, and the same segment of tape, in another playing through the tape, but this time only a vague movement without awakening occurred, with a lot of artifact in the hand channel. His GSRs have returned during sleep and are becoming irregular in latency. The small artifact in the hand channel after, "Hugh, Hugh" contributed to the score in equation (b) and was probably caused by pulling on leads due to a deep respiration associated with the large, polyphasic K-complex.

PLATE LXXII

FIG. 6.—The same subject as Fig. 5 (different session). He has been asked to respond not only to his own name but also to David, which causes brief arousal and a hand squeeze (just visible during the second half of the GSR).

FIG. 7.—An example of the scoring of the EEG responses in terms of K and K+. The GSRs which follow names played forwards may also be noted (Subject G. G. of Fig. 8).

PLATE LXXIII

FIG. 9.—To illustrate the depth of sleep in which this man, subject A. W. of Fig. 8 showed, by his GSRs, discrimination between names played forwards and backwards. He was here, of course, too deeply asleep for K-complexes to appear and his record is dominated by $\frac{1}{2}$-2 c/sec. waves with some faster spindles (Loomis D stage). His personal EEG sleep record was of rather lower than usual voltage and the fact that, as mentioned in the text, a time-constant of 0·03 sec. had to be used, should be borne in mind. His deepest sleep occurred between the 60 and 112th names of the first playing of the tape and in this period there were 30 GSRs of 2–5 cm. latency (1·5 cm. =1 sec.) to forward names, compared with 6 to backwards names. When the tape was reversed the same particular length of tape now evoked 12 GSRs of the same latency to forwards names and only 8 to the names which were now backwards, showing that loudness could not be responsible for the difference illustrated.

FIG. 10.—The subject is very deeply asleep with high voltage 1 c/sec. waves in his EEG and the swinging changes of palmar skin potential which sometimes occur in very deep sleep. He has failed to respond to the last four of the names to which he was supposed to respond—Ian and David. There has been no movement artifact for sixteen minutes. Now Ian and David come together and movement artifact is visible. No more movement occurred till ten minutes later, when the double names began.

(9)

Reprinted from *Ciba Foundation Symposium* on *The Nature of Sleep*, 1961, pp. 343–348 (edited by G. E. W. Wolstenholme and Maeve O'Connor)
Published by J. & A. Churchill Ltd., 104 Gloucester Place, London, W.1.

CORTICAL FUNCTION DURING HUMAN SLEEP

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CORTICAL FUNCTION DURING HUMAN SLEEP

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As long ago as 1830 Burdach wrote that a man did not wake up to an auditory stimulus simply on account of its intensity but on account of the personal significance which it had for him. Freud and Pavlov held similar views, and in more modern times Adrian (1937) and Bremer (1954) have both used this argument to support the view that during sleep cortical discrimination can occur, with subsequent signals descending to the brain stem, so causing more generalized arousal. A woman asleep, with, perhaps, motor cars passing by and repeatedly making a noise, and the wind repeatedly rattling the door, may wake up if someone calls "Mummy" or her name. This, however, should not be taken as evidence of a discriminative function of a complex nature such as might involve the cortex—for it is a simple discrimination between something that has been repeated over and over again and something that is new. Sharpless and Jasper (1956) sounded a given tone over and over again to sleeping cats until they were no longer aroused by it; then if they sounded a slightly different tone the cats would wake up. This discrimination was still possible in cats who had had their auditory cortex destroyed. If, however, complex patterns were used, the cat with the auditory cortex destroyed could not so discriminate. Diamond and Neff (1957) in rather clearer experiments showed also that for discrimination between complex auditory patterns an intact auditory cortex is necessary in the cat.

In experiments done at Oxford (Oswald, Taylor and Treisman, 1960) speech sounds were used. These are complex auditory

stimuli, and the clinical evidence is that one needs a functioning cerebral cortex for their discrimination. We had to consider two things: the complexity and the degree of novelty. We used a long series of spoken names recorded on tape and the subject was given the task of picking out two particular names from all these others whilst he was asleep. The degree of novelty of any particular name was no greater than the degree of novelty of the other names. A list was drawn up of 56 common Christian names and we spoke these into the tape-recorder in alternating male and female voices. We went through these 56 names again with the male voice taking the female part, and then we went through this list again and again in various random fashions. The intervals between the names were systematically irregularized so that one would not get cyclical conditioning of arousal, such as has been postulated by Pampiglione and Ackner (1958).

The subjects were sleep-deprived for 36 hours, and their EEGs were recorded, together with potentials from their hands and arms. They were asked to clench a hand when, in their sleep, their own name was called, or when one other name was called. The other name was so chosen that it had no particular significance for the given individual but so that it was the name of one of the other subjects. It was possible to control in this way for any special arousing qualities peculiar to the physical properties of a given name. The names were played by tape-recorder at about conversational level of loudness and began when the subjects were still awake, going on and on while they fell asleep and while they remained asleep.

Some of the subjects showed a remarkable capacity to respond to their own name by clenching a hand and we compared the frequency of movement in the period following each crucial name with the number of movements detectable on the record in the period of the preceding ten names. There was an enormous, and statistically highly significant, excess of movements after their own names. This also applied to the other name which was not their

own, but they could not wake up to that quite so well as to their own names; there was a statistically significant difference between the two, that is to say they moved more often following their own name than following this other name to which they had been asked to respond. Some were co-ordinated movements and some were not—some may have been chance but we had to count them all and use a statistical method.

It could be argued that this result does not bear upon the problem of whether the cortex is involved in the discrimination, because it could be said that the non-specific noise qualities, possibly coming through collateral afferents, excited the reticular formation and so caused a very brief arousal which may be manifested in the human as a K complex. Subsequent to this, with increased cortical facilitation during the latter part of the K complex when there is fast activity, the subject might examine some immediate memory store, the mechanisms of which might be independent of the level of wakefulness. The EEG data were therefore also studied. On the top of our records was a signal which indicated that a name had been called, and on the bottom of the record was written the particular name that had been called. To decide whether or not an EEG response occurs is a subjective judgement and one must rule out subjective bias. So we had a special arrangement whereby an observer scored the records with the bottom part covered over, so that he could see the signal indicating that a name had been called, and could see the EEG, but could not tell which name had been called. The EEG record was then scored for the presence or absence of a K complex response after each name.

The frequency of K complexes occurring after the individual's own name was compared with the frequency after the immediately preceding names and the results again were statistically significant. The K complex response to the subject's own name was seen in 73 out of 110 instances. The frequency of K complexes in response to the name immediately preceding was 37/110. The difference is significant ($P < 0{\cdot}001$). Only the C and D

stages of sleep were considered as K complexes do not occur in the E stage.

In addition, a subject's own name was significantly more likely to provoke a K complex than the control name. A person's own name was also more likely to evoke a K complex even in those cases where movement did not subsequntely occur—we checked on this because it was possible that on occasion the observer scoring the record was getting cues from the record when subjects woke up and moved about following their own names.

We had thought perhaps the males would wake up more to a female voice and that females would wake up more to a male voice. In fact this was not so, and they all tended to wake up more to the female voice.

We noticed that not only did the K complexes appear more often in response to the important names but they appeared to be bigger and better K complexes! Consequently we carried out a different series of experiments to check on this. We wanted also to get further data on the original problem of whether during sleep there was some process which maintained a constant "scrutiny" of incoming auditory input, and which might perhaps reject material that was not likely to be important. So we presented the sleeping subjects with names which, by means of a special technique, were played forwards and backwards, so that they would have identical stimuli, in the one condition meaningful, in the other meaningless. We compared the frequency of K complexes to forwards and backwards names under controlled conditions. The forward-played names were enormously more evocative of K complexes than the backward-played names.

A special category was used for any K complex which was a polyphasic one with three or more big slow waves. The results showed that, given that a K complex occurred at all, then it was significantly more likely to be a polyphasic K complex after a forward-played name than after a backward-played name—it was twice as likely to be polyphasic in the former case.

It would appear from these results that complex auditory discriminations can occur in the sleeping human subject (in sleep as deep as, or deeper than, the C stage) prior to arousal as manifested by the K complex.

In addition the galvanic skin responses were recorded. When subjects were awake these responses habituated to zero, but the stimuli were going on ceaselessly and, when the subjects went to sleep, in some the responses came back. Galvanic skin responses were seen to occur far more often after the forward-played names than after the backward ones during sleep.

It may be pointed out that although these backward and forward-played names were identical total physical stimuli their pattern of loudness distribution in time had been altered. A name may be loud and abrupt in its onset and quiet and gradual in its offset. Something that comes on gradually is likely to be less arousing than something that comes on sharply. We therefore carried out experiments using patterns of tones of increasing loudness, alternating with similar patterns of decreasing loudness, the total duration of each being half a second. These were played at seven-second intervals and it was shown that this loudness distribution factor, with stimuli of this duration, could not account for our results.

The experiments support the view that the sleeping human brain can carry out discriminations of such a degree of complexity as to render it highly likely that the cortex continues to function during sleep, and that exciting signals from the cortex to the reticular formation play a major rôle in arousal.

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Reprinted from the BRITISH MEDICAL JOURNAL, *May* 14, 1960, *vol. i, pp.* 1450–1455

(10)

FALLING ASLEEP OPEN-EYED DURING INTENSE RHYTHMIC STIMULATION

BY

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If one looks at translations of Russian literature in the field of medical psychology one frequently cannot but feel rather lost, for such papers are often couched in terms of the writings of Pavlov.

Pavlov's concepts are not directly compatible with Western neurophysiology, yet, since it is held that those concepts are a guide to therapy and to successful indoctrination, and since, indeed, they have been adopted by Sargant (1951, 1957) to explain sudden political and religious conversion, it is necessary that we should examine Pavlov's writings in order to reconcile them with our own neurophysiological system of knowledge. A partial attempt is made to do so here because the experimental observations with humans to be reported are closely similar to some of the phenomena Pavlov observed in his dogs, and which he believed were manifestations of "internal inhibition" in the cerebral cortex. The "external inhibition" of which he also wrote need not here concern us—it referred to the disorganization of the task in hand by an unexpected and distracting stimulus.

Internal Inhibition

Pavlov was chiefly interested in internal inhibition: "I shall call it simply inhibition, without the adjective, although each time implying internal inhibition" (Pavlov, 1955, p. 232). This inhibition was evoked in the cerebral cortex by any sensory stimulus to which, from the point of view of the dog's general economy or well-being, it was better that the dog should not respond. Throughout Pavlov's writings we find one recurring clue to the nature of this inhibition which it is possible for us to relate to our own system of knowledge—"internal inhibition and sleep are fundamentally one and the same process" (Pavlov, 1928, p. 307). "We observed that as soon as we applied the inhibitory stimulus, a somnolent state of the animal, in the form of drowsiness or sleep, immediately intervened" (Pavlov, 1955, p. 372) . . . "anyone that makes a thorough study of them will be convinced that inhibition and sleep are one and the same phenomenon" (Pavlov, 1955, p. 375).

Transmarginal Inhibition

One means by which the state of inhibition could be produced was by exposing the dog to certain stimuli which would have evoked a response had they not been excessively intense—"such conditioned stimuli too strong to give the maximal conditioned reflex, Pavlov termed transmarginal or supramaximal" (W. H. Gantt, in his introduction to his translation of Pavlov, 1941, p. 14).

Sargant (1951, 1957) interpreted human reactive collapse, after intense mental tension or excitement, in terms of the "transmarginal inhibition" caused by these transmarginal stimuli. This inhibition was held to be protective, and to be manifest in its effect on behaviour by three distinguishable phases, the "equivalent," "paradoxical," and "ultraparadoxical." These appeared when, respectively, all stimuli, whatever their strengths, acted equally; when only the weak stimuli had any apparent action; when the previously elaborated inhibitory agents alone had a *positive* effect. Pavlov wrote of this last, ultraparadoxical, phase as follows: "*In certain stages of drowsiness* [my italics] in normal dogs there occurred a distortion of the effects of conditioned stimuli. The positive stimuli lost their effect, but the negative became positive" (Pavlov, 1928, p. 345). Finally, "after this follows a state of complete inhibition" (Pavlov, 1928, p. 347)—that is, sleep supervened.

It is apparent that not only did Pavlov identify internal inhibition, as most often produced in his laboratory, with sleep, but that the variety he called transmarginal inhibition was believed by him to be of a similar nature

Human Internal Inhibition

In some experiments, described elsewhere (Oswald, 1959), in which electroencephalographic and other physiological variables were recorded, it was found, with larger numbers of human volunteers than it has been possible to use in the experiments to be reported below, (*a*) that signs of sleep appeared in persons subjected to repeated strong electric shocks, (*b*) that signs of sleep could come and go rhythmically in time with regular stimuli at intervals of only a few seconds, (*c*) that signs of sleep appeared while subjects continued to move in time with prolonged, rhythmic music. It is obvious that condition (*a*) could be labelled "transmarginal inhibition," and Sargant (1957) laid great emphasis on the use of prolonged movement, to rhythmic music, as a means of inducing this state of inhibition, quoting with approval the view of Hecker that the state induced is "like that of small animals when they are fascinated by the look of a serpent." This latter condition of fascination, or "animal hypnosis," was shown by Gerebtzoff (1941), and others subsequently, to be electroencephalographically a state of sleep. However, in the experiments with human volunteers mentioned above, the eyes were always closed. Would comparable signs of sleep appear under such circumstances in persons whose eyes were open?

The example borne in mind was that of a prolonged tribal dance where not only does the individual move in time with the rhythm of the auditory stimuli, so

provoking bodily sensations occurring at the same rhythm, but he is stimulated visually, at the same rhythm, by the sight of others moving.

In other experiments, I have encountered some persons who would sleep on undisturbed when their eyes were pulled open during deep sleep beneath a strong light. Again, children may not infrequently be seen asleep with their eyes partly open, particularly during febrile illness. However, it is one thing to be undisturbed by visual stimuli when already asleep, another actually to fall asleep with the eyes open during visual stimulation.

Signs of Sleep

While the most reliable signs of sleep in a normal person are probably electroencephalographic, among other firmly established signs are slowing of the heart (Boas and Goldschmidt, 1932) and pupillo-constriction (Kleitman, 1939 ; Byrne, 1942).

The electroencephalogram (E.E.G.) of the average alert person whose eyes are open tends to be flat in appearance, with some low-voltage fast waves. If he relaxes with eyes closed, the alpha rhythm, at about 10 c./sec., may appear, but it will do so also if he is bored while his eyes are open, and this is probably related to specific visual inattention. As the individual drowses, the alpha rhythm slows and disappears, the E.E.G. record becomes rather flat again, but with low-voltage slow waves at 4–6 c./sec. As sleep deepens these slow waves are accentuated, and, in many persons, bursts or spindles of faster waves at about 12–15 c./sec. are recorded from the anterior part of the scalp. This last state is one of medium-depth sleep. The stage of a flat E.E.G. record with low-voltage slow waves is generally considered to be sleep, and is the stage, above all others, for hypnagogic hallucinations and dreams ; indeed, Dement and Kleitman (1957) maintain that dreams occur only in this stage. The transition from wakefulness to sleep is a gradual and continuous, not an abrupt, discontinuous process.

The Experiments

It was planned that the whole environment of each volunteer should be overwhelmingly (or "trans-marginally") dominated by one major rhythm of stimulation. A 60-minute tape-recording of non-stop "blues" music, with the recording on the upper track of the magnetic tape, was played, and on the lower track of the tape there was recorded by hand a sequence of brief 800 c./sec. tones, which went on and off with each succeeding beat of the musical rhythm. A Brenell Mark V twin-track tape-recorder was used for this. In subsequent experimental sessions both tracks of the magnetic tape were played simultaneously, the upper one into a loudspeaker, and the lower one into a transistorized device containing a high-speed relay which closed during each 800 c./sec. tone (which was inaudible, of course). This relay, in turn, was used to actuate other relays which (*a*) switched on and off the mains supply to the visual stimulator (see below), and (*b*) could deliver repeated electric shocks—in both cases at a rhythm *synchronous* with the rhythm of the music.

The Visual Stimulator.—Visual stimulation was provided by four 60-watt electric-light bulbs, one at each corner of a 2 by 2 ft. (60 by 60 cm.) piece of hardboard, which was otherwise featureless except for a couple of brass screwheads in the centre.

Electric Shocks.—These could be provided from a 0.05-microfarad condenser, rhythmically charged to 300 volts, and rhythmically discharged, through the leg of the volunteer, by means of a double-pole break-before-make relay. A 20-kilohm potentiometer, in series, could be used to adjust the severity of the shock.

The Eyes.—The volunteer subjects' eyes were fixed open throughout the experiments, during which a kettle was kept boiling in the room to minimize corneal drying. Rapid-drying collodion was applied, through a fine nozzle, to a narrow strip of the upper eyelid, about 2 mm. from the margin of the eyelashes. The end of a strip of half-inch (1.25 cm.) adhesive tape was applied to the collodion, and further collodion was placed along the line of contact just clear of the eyelashes. When the collodion had dried, the eyes were opened and the upper eyelid was fixed by attaching the free end of the adhesive tape to the forehead with sufficient tension on each side to prevent the lights of the visual stimulator disappearing from the field of vision, even when the subject voluntarily rolled his eyes upwards to the greatest possible extent.

Volunteer Subjects.—They were paid volunteers who were physically and psychologically normal. They had each, in the past, been subjects in experiments involving electroencephalography, and both their waking and sleeping E.E.G. records were known to be unremarkably normal. Three of the subjects worked in an electroencephalography department and one other was an Oxford undergraduate. Needless to say, they had no history suggestive of narcolepsy.

Experiments with Subjects Immobile

Three volunteers were used in these experiments. The subject lay on a couch with the visual stimulator about 2 ft. (60 cm.) above his face, which meant that there was a peripheral visual field containing, among other detail, equipment, curtains, windows, and myself. Stick-on silver electrodes on the scalp (Fig. 1), and arms, recorded the E.E.G. and E.C.G. Two further electrodes, for electric shocks, were placed on the left leg, one over the lateral surface of the neck of the fibula, in order to stimulate the lateral popliteal nerve fairly directly, and a further one over the anterior surface of the tibia a couple of inches away. The passage of the current between these two electrodes stimulated the lateral popliteal nerve sufficiently to cause a sharp eversion of the foot with each shock, so providing rhythmic proprioceptive as well as rhythmic and sharply uncomfortable skin sensations synchronously with the musical rhythm and the light flashes. The music was always very loud.

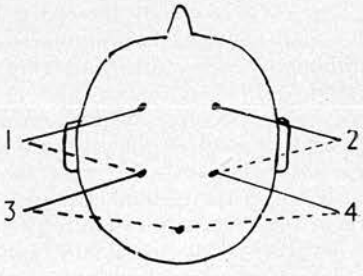

FIG. 1.—Showing positions used for E.E.G. scalp electrodes. The numbers 1–4 in subsequent figures refer to the positions shown here.

Subject A

This subject was a healthy 23-year-old university-educated man who worked in an E.E.G. department. He was very athletic, and his heart rate, when awake at rest, was only 40/min., so that his heart rate of 36–40/min. when asleep did not provide useful information. As this was the first

attempt to observe someone fall asleep with open eyes, this one subject was used when deprived of sleep, after less than one hour's sleep the previous night. The experiment was done at 3 p.m. in a shaded room.

Though accustomed to electroencephalography and rhythmic music, he had never before received electric shocks deliberately, nor the flashing lights. Despite this, his E.E.G. showed him to be lightly asleep after 12 minutes, when there was also some eye-rolling, and he remained asleep (Fig. 2)

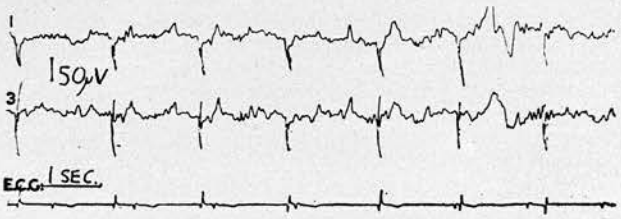

FIG. 2.—Subject A. Eyes glued open during loud music, rhythmic light flashes, and electric shocks. He rapidly went to sleep, characterized by slow waves and 14 c./sec. spindles in his E.E.G., as illustrated. The large spike artifacts in both the E.C.G. and E.C.G. channels occurred as the lights went off; half-way between each of these a small artifact is visible in the E.E.G.—the moment of the shock and the oncoming of the lights.

for the next half-hour, with only intermittent arousal, due to faults in the apparatus which sometimes caused a double electric shock with a consequent break in the rhythm. The session was then ended. His pupil had been noted to be very small while he was asleep, during most of which time he eyes remained motionless in what appeared, from an oblique view, to be the mid-position.

He declared afterwards that he was aware of having been asleep and of once thinking, "The shocks have stopped," and then realizing that, in fact they had not, but that he had been asleep.

Subject B

This subject was not sleep-deprived, and, though the experimental session was held between 9 and 10 p.m., this was several hours before his usual bedtime. He was a 24-year-old Oxford undergraduate, reading psychology and philosophy, a big, hearty, athletic ex-naval officer.

He had been used as a subject in some quite different E.E.G. experiments some months previously, but had never experienced stimulation of the kind used in the present experiments, and complained more about the electric shocks than did the other subjects. As it was evening, the general room illumination was low (one 75-watt bulb 10 ft. (3 m.) from the subject) and the four electric bulbs before his eyes were so bright that at first he declared he could not possibly stand their glare. However, he was assured that he would get used to it in time—and he did. Fig. 3 illustrates the course of events, from the flat E.E.G. record of alertness, through a stage of diminished alertness with return of alpha rhythm, to a stage of drowsiness with low-voltage E.E.G. slow waves (after eight minutes), to unequivocal sleep after another two minutes, though with only a small fall in heart rate, from 72 to 66/min. While he was asleep his pupils were seen to be small, and, seen from an oblique view, appeared central. He was afterwards fully aware that he had been asleep.

Subject C

This subject, a healthy 20-year-old man, worked in an E.E.G. department, and had, some two years previously, received electric shocks in the course of other experiments. He was in no way sleep-deprived, and the experiment was conducted during the afternoon in a shaded room.

The course of events was similar to that with Subject B. He was asleep within eight minutes (Fig. 5), and after a further 15 minutes was aroused by a double shock (caused by an apparatus fault) which destroyed the rhythm. After this he remained drowsy for another 15 minutes till the session ended. During much of the latter period his basic E.E.G. record, as it were, was that of light sleep—that is,

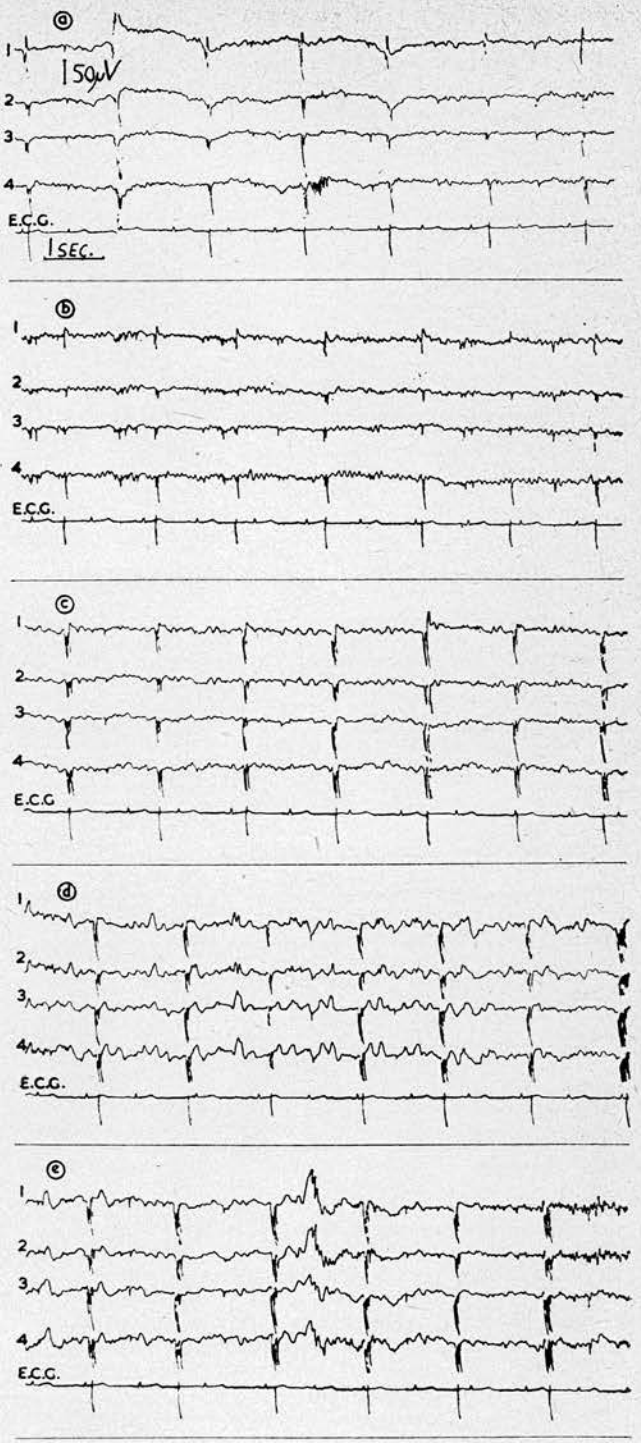

FIG. 3.—Subject B. Eyes glued open. Rhythmic electric shocks and lights. Large artifacts at time of lights off, small artifacts at time of lights on and shock.

(*a*) Top excerpt shows E.E.G. appearance after 1 minute. Note flat record with low-voltage fast waves—the normal appearance in a waking person with eyes open.

(*b*) Second excerpt, after 4½ minutes. Over the back of the head (channel 4 especially) alpha rhythm has reappeared even though the subject's eyes are open.

(*c*) Third excerpt, after 8 minutes. The E.E.G. appearances are now those of very light sleep. The alpha rhythm has disappeared and low-voltage slow waves can be seen.

(*d, e*) After 8 minutes the subject is asleep (bottom two excerpts) and the E.E.G. record is dominated by slow waves and occasional K-complexes appear (the big slow wave followed by 12-c./sec. waves) and anterior 15-c./sec. sleep spindles (channels 1 and 2). His pupil was by now very small.

flat with low-voltage slow waves—but each light flash tended to cause him to alternate between three-quarters of a second in light sleep and three-quarters of a second in a more aroused state in which alpha rhythm was present (Fig. 6).

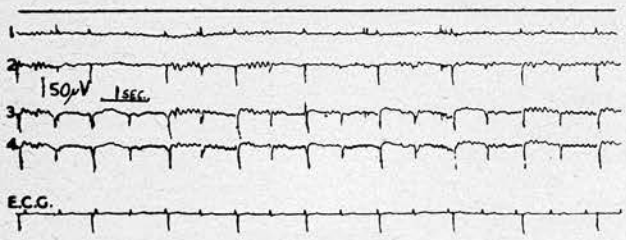

FIG. 4.—Subject C. Eyes glued open, rhythmic music, electric shocks, and light flashes. After three minutes the E.E.G. record is no longer that of continuous full alertness. The alpha rhythm may be seen, in channels 3 and 4, to appear at times when the lights are off. The big artifacts indicate lights off, the smaller ones indicate lights on. While the lights are on, some 50-c./sec. mains interference occurs.

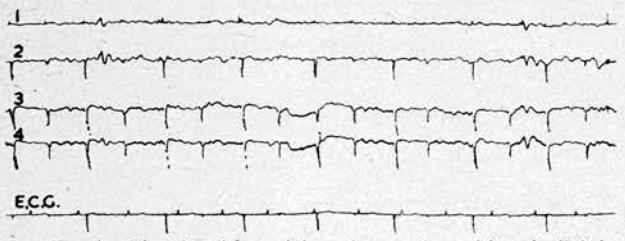

FIG. 5.—As Fig. 4. After eight minutes the subject is lightly asleep and his E.E.G. record is one of low-voltage slow waves. Occasional 12-c./sec. sleep spindles (not shown) also occurred.

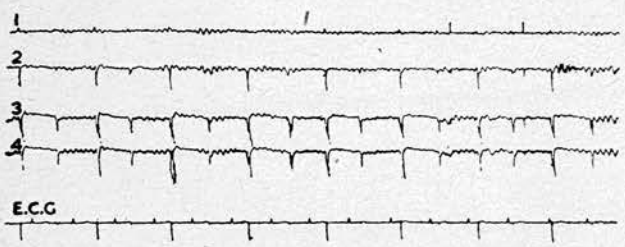

FIG. 6.—As Figs. 4 and 5. The subject was aroused by an unintended double shock, and has drowsed since; in contrast to Fig. 4, his alpha rhythm now tends to return when the light is on (during the thick, heavy 50-c./sec. artifact), which rhythmically brings him from, as it were, a state of light sleep to one of low-grade wakefulness.

This was in contrast to the first few minutes of the session, when the light had tended to make him alternate between a very alert (light on, no alpha rhythm) state and a less alert (alpha rhythm present) state (Fig. 4).

While his E.E.G. showed signs of sleep, his pupils were seen to be small and rolled upwards, though not sufficiently to obscure the pupils. His heart rate, which had been 80–85/min., fell to 55–65/min.

He subsequently reported that he was quite aware of having been asleep for part of the session.

Experiments with Subjects Actively Moving

The experiments already described involved rhythmic leg movement to the music, but this was really passive movement caused by the electric stimulation. Two further sessions were undertaken in which subjects did not receive electric shocks but actively banged both arms up and down from the elbows, while sitting up in a chair, and tapped both feet in time with the rhythm of the music and the flashing lights.

The visual stimulator was again positioned about 2 ft. (60 cm.) in front of the eyes, which, as before, were glued and strapped fully open. Both sessions took place during the afternoon, neither subject was sleep-deprived, and the room was shaded but not darkened.

Subject C

In the first few minutes his E.E.G. was the fairly flat record characteristic of full alertness with the eyes open, though within seven minutes his alpha rhythm had become fairly continuous and a little slow activity was beginning momentarily to appear. After 10 minutes there came the first break in movement lasting three seconds, and this was associated with the E.E.G. slow-wave picture of light sleep. In the next 15 minutes there were 65 episodes, lasting 3–20 seconds, of failure to move in time to the music, each one associated with E.E.G. signs of light sleep and a considerable slowing of the heart (see Fig. 7). In these episodes of light sleep his eyes were rolled upwards, but not enough to obscure the pupils.

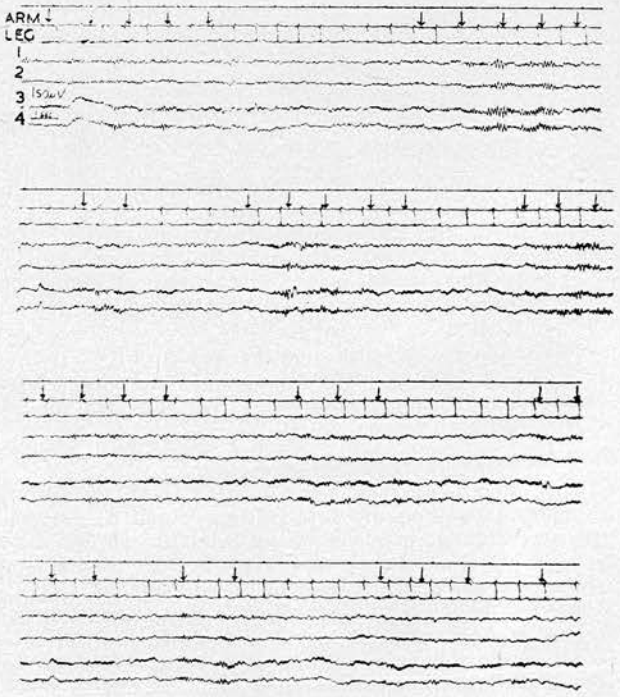

FIG. 7.—Subject C. Illustrates the frequent, brief episodes of light sleep, during which the E.E.G. alpha rhythm disappears, to give place to low-voltage slow waves, the heart slows (the E.E.G. is present in the arms and leg channels) and movement ceases. The arrows point to bursts of muscle potentials with each movement. The heavy 50-c./sec. interference in channel 3 indicates when the lights were on. The segments of record are continuous from above down. Electrodes over the forearm muscles and over the gastrocnemius and peronei show bursts of muscle spikes from the leg and arm in this and subsequent figures.

Questioned afterwards, he said that once or twice he had "come to" suddenly and realized that he might have been asleep. He had had to urge himself to keep moving. Asked if he thought he had stopped much, he said, "Yes, but not more than half a dozen times."

Subject D

This subject, a 24-year-old healthy university-educated man, worked in an E.E.G. department. He moved uninterruptedly to the music for 15 minutes, during which time his alpha rhythm gradually became more or less continuously present, after which the first break in arm movement occurred, associated with a few seconds of E.E.G. signs of light sleep. Further breaks in movement, 52 in all, each associated with E.E.G. signs of sleep and usually an obvious slowing of the heart, occurred in the next 25 minutes. Fig. 8 illustrates. The episodes of sleep were associated with small pupils and a variable position of the eyes, which would roll to one side for perhaps 10 seconds, then perhaps upwards (but still with exposed pupil) for a like period, then else-

where, and so on. The breaks in movement bore no obvious relation to the position of the eyes.

Questioned afterwards, he emphatically maintained that he had stopped moving only once. He said that, at first, luminous geometrical patterns had seemed to be present on the hardboard of the visual stimulator, but later he became unaware of seeing anything.

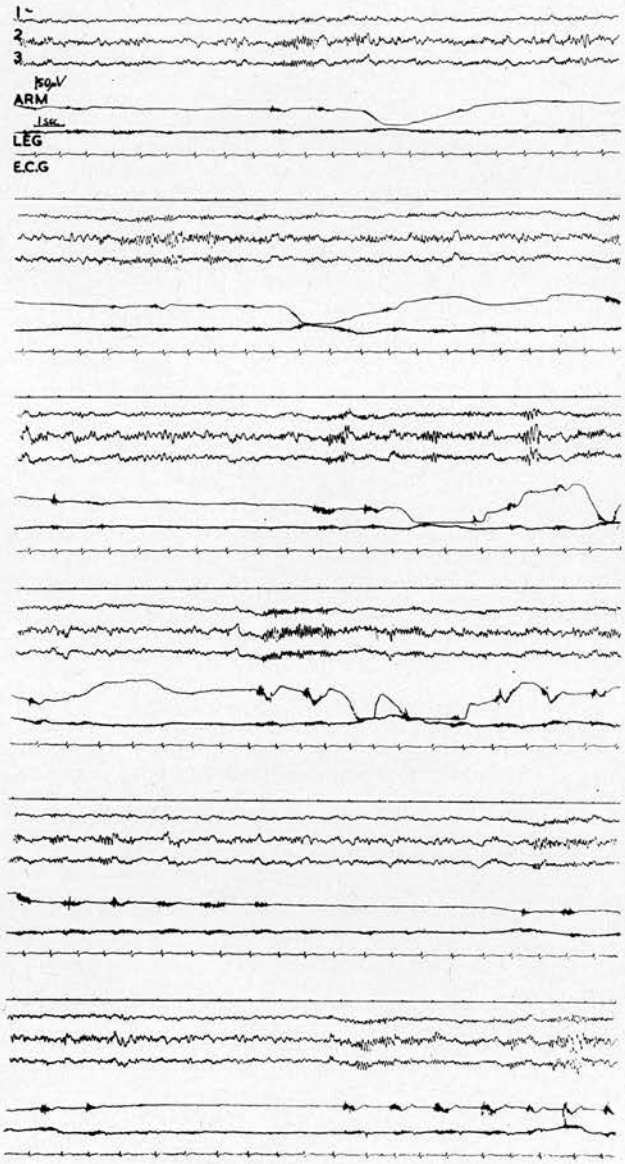

FIG. 8.—Subject D. Eyes glued open while moving actively to rhythmic music and rhythmic light flashes. Illustrating how repeated brief episodes of light sleep were manifested by slow waves in the E.E.G., some slowing of the heart, and failure of movement. Bursts of muscle spikes can be seen to persist longer in the leg than in the arm channel. Just before each return of movement, after a sleep episode, the sleep pattern changes momentarily to a low-voltage fast pattern prior to the alpha rhythm return. The segments of record are continuous. Channel 2 of the E.E.G., in this instance, derived from the front right and the nearmost electrode.

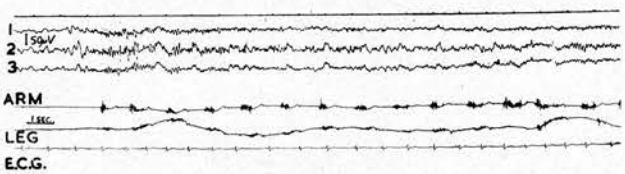

FIG. 9.—As Fig. 8, but to illustrate how, at times, movement continued when the E.E.G. record was, nevertheless, dominated by slow waves.

Compared with experiments previously conducted on volunteers with closed eyes (Oswald, 1959), Subjects C and D tended much more often to cease moving altogether during E.E.G. sleep signs, whereas subjects with eyes closed more often went on moving during similar E.E.G. signs, even though the quality of movement deteriorated. Nevertheless, as may be seen in Fig. 9, there were times when movement continued while the subject's E.E.G. contained a good deal of slow activity.

Discussion

The changes in the E.E.G. seen in the present experiments, which are those characteristic of persons falling asleep under conventional circumstances, were accompanied by slowing of the heart in three of the four subjects, and by obvious pupillo-constriction in also three of four subjects. Furthermore, the subjects in the experiments involving shocks were all quite certain they had been asleep. This was in contrast to the two subjects who moved to the music. The obvious difference between these circumstances was that in the former case subjects slept for minutes at a stretch, while in the latter case subjects appeared to sleep for only a few seconds at a time.

It seems reasonable to believe that each of these volunteer subjects did go to sleep, but it will be remembered that there is no clear dividing line between wakefulness and sleep, and it is no part of my present concern to insist that subjects crossed any such dividing line, only to claim that there was a considerable fall of cerebral vigilance, and a large decline in the presumptive ascending facilitation from the brain-stem reticular formation to the cerebral cortex.

The eyes of two of the four subjects remained fairly central, and only in one case were the eyes steadily turned upwards. Upward rotation is common, though far from universal, during sleep (Pietrusky, 1922), but Kleitman (1939) noted that if the eyelids were opened and then kept apart during sleep the eyes would sometimes return to the middle position, as I have also seen in other experiments.

The problem which led to the present experiments has already been mentioned—namely, whether intense rhythmic stimulation would produce a sleep-like condition in persons whose eyes were open, for this condition could then be related to the transmarginal inhibition of Pavlov, which Sargant believed developed as a result of intense rhythmic stimulation. The importance of this lies in the differences in the *quality* of thinking between the fully awake state and drowsiness or light sleep. We believe to-day that, other things being equal, fully efficient cortical activity depends upon an optimal, high degree of ascending facilitation from the brain-stem reticular formation. Lesser degrees of that facilitation would be associated with impaired capacity for higher-quality thinking. One of the characteristics of the latter is that numbers of more or less independent facts can be simultaneously related together—in particular, facts derived from the past can be related to current or projected activities or beliefs.

McKellar (1957) has drawn attention to the differences between thinking in the fully alert condition and thinking in light sleep—that is, dreaming—the former "reality-adjusted" thinking, and the latter illogical, "autistic" thinking, which is no longer governed by "repeated appeal and submission to established fact, whether perceived or stored" (Oldfield, 1959).

Autistic thinking in dreams, though illogical, perhaps confused, yet intensely personal, does not quite conform to the "ultraparadoxical" behaviour during drowsiness that Pavlov described, and on which Sargant drew to exemplify, for instance the new love for a formerly hated interrogator, during the state of transmarginal inhibition. Scrutiny of Pavlov's writings, however, leaves some doubt on how firmly established and clearly demarcated were the three phases he distinguished, and even more doubt on how confidently these changes in the salivary secretion of dogs, in the particular circumstances of Pavlov's laboratory, should be extrapolated to human behaviour. It would seem safer merely to recognize that in certain phases of drowsiness the dogs failed to respond in their usual way to stimuli of learned significance, and that the dogs' behaviour became illogical, disorganized, and confused.

The present experiments have illustrated the effects of extremely monotonous sensory stimulation on persons required to *keep still* or make only repetitive movements —the former factor is very important, as discussed elsewhere (Oswald, 1959). Yet there can be little doubt that changes of the same kind, if of lesser degree, must inevitably occur in persons engaged on other monotonous tasks. Apparently trade union opinion was against an unaccompanied driver for diesel locomotives, and one may be glad that the man who has to sit at the front of the train, subjected to its rhythmic chatter, the sight of telegraph poles flashing by, and the rails and sleepers disappearing endlessly below has someone else with him to provide occasional conversation—in fact, to introduce some variety, and help to prevent a state developing similar to that seen in Subjects C and D, who, though aware only occasionally of "coming to," yet went to sleep so frequently.

What physiological mechanisms underly this response to monotony? Let it be accepted that the degree of wakefulness/sleepiness depends on the level of excitement of the activating system in the brain-stem reticular formation, and the amount of consequent ascending facilitation from it to the cortex. Let it be accepted also that that activating system is kept excited by impulses arriving either directly or indirectly as a result of stimulation of sense organs. If the impulses were to become repetitively monotonous, what would be expected to happen? There is every reason to suppose that the usual response—namely, excitement of the activating system—would no longer appear, for this phenomenon of *habituation*, or cessation of response on repeated stimulation, is a property of stimulus-response mechanisms of all degrees of complexity (Humphrey, 1933; Oldfield, 1937). Characteristic of this failure of response is the immediate restoration of response, or *dishabituation*, following a change such as an alteration in the rhythm of stimulation—and in the present experiments the arousal reappeared on several occasions when an unintended double electric shock was given.

Hugelin and Bonvallet (1957) have presented evidence to indicate that cortico-fugal impulses can damp down the activating system in the brain-stem reticular formation. In the case of a person who is obliged to keep still and who knows he can do nothing about the sensory stimulation, perhaps inhibitory, "Keep still! Don't move!" signals from cortex to brain-stem might well play a part in causing sleep, as is presumably the case in the rabbit fascinated by the snake.

Summary

Pavlov wrote of the basic identity of sleep and states of inhibition caused by certain forms of sensory stimulation.

Experiments are described in which male volunteers, whose eyes were glued widely open, went rapidly to sleep during simultaneous, *synchronized*, rhythmic electric shocks, loud rhythmic music, and strong flashing lights. Active rhythmic movement to the music, in place of the shocks, also appeared to cause subjects to sleep very briefly for a few seconds at a time, often with moments of failure to move in response to the rhythmic music—moments of which the subjects were subsequently largely unaware.

The significance of these observations is discussed in the light of Sargant's proposals concerning "transmarginal inhibition" in humans, and attention is drawn to the failure of "reality-adjusted" thinking in states of light sleep, and also to the almost inevitable occurrence of similar brief episodes of light sleep in persons, such as some drivers, who are exposed to prolonged, monotonous sensory stimulation.

I wish to acknowledge the encouragement and facilities given me by Professor R. C. Oldfield, and to pay tribute to the fortitude of the volunteer subjects. Mr. P. G. M. Dawe kindly made the transistorized high-speed relay device.

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Psychological Review
1961, Vol. 68, No. 5, 360–362

ON THE ORIGIN OF THE EEG ALPHA RHYTHM

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In reporting some ingenious experiments Kennedy (1959) suggests that oscillating fluctuations of electrical potential recorded in the EEG may be artifacts resulting from the mechanical (arterial) pulsation of a charged gel in a rigid case. Kennedy confines his attention to the alpha rhythm and to a rhythm which he calls the anterior temporal rhythm, originally claimed to be associated with thinking (Kennedy, Gottsdanker, Armington, & Gray, 1948), although, to the best of my belief, independent confirmation is still lacking.

Kennedy proposes that variations in the amount of human alpha rhythm result from variations in local blood flow in the brain, as a result, he implies, of the activity of the autonomic nervous system. Brain waves were shown to vary in association with changes of cerebral blood flow by Ingvar (1955) and Ingvar and Söderburg (1956), following reticular formation stimulation, but the blood flow changes always occurred several seconds after the electrical changes, just as the peripheral vasoconstriction which follows a startling stimulus has a latency of 1–5 seconds, compared with 0.3 seconds for alpha blocking in man. It is possible, by repetitively varying what may be called "attention," to cause fluctuations between the alpha rhythm picture of wakefulness and the nonalpha picture of light sleep, or alternatively, the nonalpha picture of high level wakefulness, in an exact and rhythmic fashion, with rhythms having periods of 1–2 seconds (Oswald, 1959b, 1960). Vasomotor responses are too slow to account

for such accurate, rapid, and rhythmic alterations.

The alpha rhythm can be replaced by high voltage slow waves, not only during the cerebral vasoconstriction of hyperventilation, but also during the cortical hyperaemia and congestion of encephalitis. If a patient with slow waves owing to encephalitis hyperventilates, so causing cerebral vasoconstriction, the slow waves become more marked; they do not change to alpha rhythm during the time of some "optimum" vasomotor state.

The alpha rhythm disappears at the onset of sleep, and Kennedy quotes Shepard's work during the early years of this century to support the view that increase of cerebral blood flow causes the change of the EEG. Other workers, before and since, obtained contrary results but, using modern techniques, Mangold, Sokoloff, Conner, Kleinerman, Therman, and Kety (1955) did find evidence of a small but significant increase of cerebral blood flow. Central nervous system responsiveness is lowered in sleep, including the responsiveness of the respiratory centre to carbon dioxide, the arterial concentration of which rises during sleep (Bellville, Howland, Seed, & Houde, 1959; Magnussen, 1944). Carbon dioxide is a very potent vasodilator and it is believed that this is responsible for the increased cerebral blood flow in sleep (Robin, Whaley, Crump, & Travis, 1958). The disappearance of the alpha rhythm at this time is preceded by a *slowing* of the rhythm. Yet when carbon dioxide is inhaled during wakefulness, causing a big increase of cerebral blood flow (Kety & Schmidt, 1948), the alpha rhythm does not slow but becomes *faster* (Gibbs, Williams, & Gibbs, 1940).

Inhalation of a low oxygen–high carbon dioxide mixture sufficient to produce an enormous increase in cerebral blood flow (Kety & Schmidt, 1948) can be without effect on the EEG frequency distribution (Holmberg, 1953).

The Figure 2 of Kennedy's paper apparently shows the artificial "alpha rhythm" fluctuating in amplitude at the rate of the "pulse." There are theoretical reasons for believing that this might occur in man for quite different reasons, and I observed a case in which awareness fluctuated with the arterial pulse (Oswald, 1959a). Subsequent attempts of mine, using superimposition photography, to demonstrate fluctuation of alpha "envelope" amplitude with the pulse met with no success.

Kennedy claims that a hole in the skull will greatly modify or abolish the alpha rhythm. Although he quotes the writings of Jasper, he does not mention the recording of alpha rhythm from the exposed brain of the conscious human, shown by Penfield and Jasper (1954, p. 187). Kennedy's crucial experiment remains unconvincing, for he shows us but two selected parts of the EEG record from his subject, in each of which only a couple of seconds of eyes-closed record are shown. Had he presented hundreds of such examples, completely unselected, to an independent observer denied all knowledge of the presence or absence of the "damping mechanism," that observer could then have made judgments as to the degree of alpha rhythm present, and we might have been in a better position to judge the reliability of the phenomenon in question.

According to Kennedy the alpha rhythm should be extremely sensitive to changes of cerebrospinal fluid pressure. A simpler crucial experiment lies in the examination of the effects of variations of this pressure in normal people. Over a number of years, and for a variety of reasons, I have studied alpha rhythms from the same individuals in both the upright and the prone positions and have never noticed any difference in the alpha rhythms, despite the fact that the cerebrospinal fluid pressure within the skull varies considerably with posture. A further simple, deliberate means of testing Kennedy's hypothesis was provided by jugular vein compression (Queckenstedt's maneuver) and also forced expiration against a closed glottis. These procedures both cause a sudden, large rise of cerebrospinal fluid pressure within the skull. These maneuvers, when first attempted with two normal subjects, caused alpha blocking, but when they were re-

peated half a dozen times, so that the subjects became used to them, no change of alpha rhythm was to be seen.

It would be of interest to learn Kennedy's views on the "following" of the human occipital EEG rhythms at the frequencies of a flickering photic stimulator. In some persons these rhythms may follow faithfully the frequency of the flicker from 2 to 20 cycles per second.

If I follow Kennedy correctly he does not imply that all EEG waves could be attributed to the phenomenon he has demonstrated. Indeed I suspect he would be hard put to it to explain the vast quantity of observations made in recent years on the brains of cats lacking major portions of their calvaria, or that most striking feature of the human EEG during medium depth sleep, namely the K complex, with its short latency and composite pattern of slow and fast waves following a sensory stimulus. It would then be necessary to claim that the human alpha rhythm is a special case.

As I fail to find Kennedy's arguments convincing, I shall continue to believe that the alpha rhythm comes and goes in relation to increased alertness (Oswald, 1957) and especially visual alertness (Oswald, 1959c) on the one hand, and light sleep on the other, by reason of mechanisms which embrace EEG phenomena as a coherent whole.

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(Received August 16, 1960)

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INDUCTION OF ILLUSORY AND HALLUCINATORY VOICES WITH CONSIDERATIONS OF BEHAVIOUR THERAPY

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Reprinted from
THE JOURNAL OF MENTAL SCIENCE
Vol. 108, No. 453, March 1962

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Introduction

In recent years interest has attached to the fact that experiences allied to those described by patients during acute schizophrenic illnesses can be induced in normal people by the use of certain drugs, sometimes called "hallucinogens". In so far as these drugs have caused hallucinations, however, they have induced predominantly *visual* hallucinations, whereas in schizophrenia the outstanding hallucinations are *auditory* ones, of voices—often making remarks in the third person singular, sometimes making apparently senseless or absurd remarks. In this paper, attention is drawn to means whereby non-schizophrenic persons can be caused to experience hallucinatory and illusory voices by primarily psychological and not pharmacological techniques. These techniques are also applicable to normal volunteers, but this paper will deal with first experiences of them during the treatment of some sexual deviants and alcohol addicts.

The auditory experiences to be described include easily understandable illusions or mishearings which, however, develop from previous veridical perceptions and which occur when the individual has repeated opportunities to "correct" his erroneous perception. At the other extreme are frank hallucinations accompanied by other disturbances of a kind characteristic of schizophrenic, dereistic, or "primary process" thinking.

While it is customary to draw sharp distinctions between illusions, images, pseudo-hallucinations and hallucinations, I believe this to be dictated by the practical needs of communication, and that to do so may create false divisions in our understanding. They are all percepts which depart more or less markedly from that correspondence which customarily exists between percept and sense-organ stimulation.

Illusions are commonly held to be percepts deriving from the "misuse" of information supplied by the sense organs. There is an implicit assumption that the exteroceptors are inactive except when directly stimulated, whereas we now know that sense-organ receptors maintain a resting rate of discharge, the *pattern* of which becomes altered by direct stimulation. In the absence of obvious visual stimulation, for instance, a heavy rate of discharge of retinal receptors still occurs, so that we could, if we wished, argue that all visual hallucinations are illusions. Indeed for over a hundred years it has been argued that hypnagogic visions are illusions based on the idio-retinal light or *eigenlicht*.

The distinctions between images, pseudo-hallucinations and hallucinations refer to the degree to which the individual is capable of relating his experiences to total reality past and present. It is submitted that there are not normal and abnormal images so much as that there are normal and abnormal possessors of images. Those who tend to experience vivid visual imagery during recall are usually unjustifiably confident of its veridical nature. Dream percepts may be rejected, on subsequent waking reflection, as unreal, and termed pseudo-

hallucinations, but at the time they were experienced there was loss of contact with reality and they were accepted, in fact they were hallucinations. Similarly some psychotic patients may, on recovery, reject their former hallucinations as unreal. The difference lies in the length of time which elapses prior to that rejection and not in the nature of the experiences themselves, nor, necessarily, in the neurophysiological correlates of those experiences.

Outside psychiatric practice, illusions and hallucinations can occur in the hypnotic trance, but otherwise occur under two main kinds of conditions, or a mixture of both. First, when discriminative efficiency is impaired by sub-optimal brain function—examples are conditions prevailing in sleep, fever, and following various toxic agents. Hallucinations are common in the recurrent momentary drowsiness of sleep-deprivation and hallucinatory voices often occur in normal drowsiness (Oswald, 1962) when there is also "sensory isolation".

Second, when the normal perceptual cues are diminished or are ambiguous. Hallucinations are common in "sensory deprivation" or "sensory isolation" where contact with present reality is grossly attenuated; under these circumstances, some degree of diffuse light favours visual hallucinations (Cohen *et al.*, 1959)—an obvious instance where the term "illusions" could as easily be applied. If the individual, by repeated experience of a certain class of percept, is led to expect further such percepts, then he may hallucinate what he does not reject as unreal because it was fully expected—Mowrer (1938) has reported apparent faint tactile hallucinations, Ellson (1941) auditory hallucinations of very faint tones, and Oswald (1959a) hallucinations of severe electric shock and powerful light flashes, induced by these means.

The so-called experimental "sensory deprivation" is never absolute and, since sensory receptors respond especially to change of stimulus, and since rapid habituation of central responses occurs when stimulation is monotonous, the situation is essentially one of environmental uniformity. In the observations described below, the gross artificiality of the usual experimental "sensory deprivation" was not present, but there was an imposed uniformity of all that was really *significant* for the individual. Lindsley (1961) interpreted the phenomena of "sensory deprivation" in terms of the physiology of the ascending reticular formation of the brain-stem. While the original supposition was that the latter was kept excited by any sort of afferent inflow via collateral paths, it can be argued that more important in man are cortico-fugal impulses to the reticular formation consequent upon stimuli *significant* for the individual, the action of which it has been possible to demonstrate (Oswald *et al.*, 1960).

Method and Results

Seven patients were concerned in this work. Each received a course of aversion therapy in which they had two-hourly injections of 1/10 grain apomorphine hydrochloride, with occasional rest periods. Some also received occasional pilocarpine nitrate injections, and some small doses of dexamphetamine. It will become clear, however, that the phenomena were not contingent upon dexamphetamine. The vomiting naturally produced electrolyte depletion, but this alone did not provoke, though it may have favoured, the auditory hallucinations, and the absence of vomiting did not prevent the response in Case 7. The critical factor determining auditory misperception was repetitive verbal stimulation.

In each case a loop of magnetic tape was made by joining the cut ends of

a length of tape. On each loop remarks of calculated personal significance to the patient were recorded and it was played by a modified tape recorder, so that the same loop went round and round endlessly.

Patients' reports of their experiences were tape-recorded, while in some instances they wrote down what they heard at the time.

The cases are presented in their actual temporal order. The auditory experiences the patients had were unexpected. The first case was actually the most dramatic, and because of this and the therapeutic success, it is presented in detail. The remainder are much abbreviated.

Case 1. We shall here give his first name as Jim and, since it is important to state that his surname is Brown, we shall otherwise mention only that he was aged 22 and well-educated. One maternal grandmother had a mental disturbance during senility and his father is said to have been depressed (though not under treatment) during a period of unemployment in the 1930s. Otherwise there was no personal or family history of mental illness.

He was a rubberized-mackintosh fetishist. He recalled an incident involving rubberized cloth when aged 3. Since the age of 7 or 8 he had been sexually excited by the sight of women wearing rubberized mackintoshes and by the age of 10 was masturbating frequently to the accompaniment of fantasied or actual mackintoshes. He had married 8 months prior to coming for treatment and had normal heterosexual desires, except that he frequently wished his wife to wear her rubberized-mackintosh in bed. He occasionally asked her to do so, she refused, and they both became distressed. He admitted no sado-masochistic or other abnormal interests. In view of the eventual success of treatment, it should be made clear that, apart from the fetishism, he was the only case in this series who could be said to be of a really normal personality.

He was admitted for a course of aversion therapy. Several rubberized-mackintoshes were hung from the walls and every two hours, after each injection, he stripped, donned a mackintosh, lifted his wife's mackintosh from off a bowl and vomited into the bowl. At the alternate hours copious orange drink and milk were given to combat fluid and electrolyte depletion. By the third day he was aggressively rejecting the clothing and at noon received, as an extra, 1/10 grain of pilocarpine nitrate by injection because the vomiting was decreasing. On the first two nights he twice received 10 grains of dexamphetamine sulphate by injection.

On the third day, because the routine seemed too much like a laboratory exercise divorced from his personal relationships, a tape-loop was played continuously from 3 p.m. onwards. It ran as follows:

A faint male voice (mine) saying, "Rubberized clothing makes . . ." "Rubberized clothing makes him sick" (female voice). The noise as of a man vomiting and then female laughter. "Rubber mackintoshes make him sick" (a second female voice). Vomit noise. Female laughter. Three clicks on the tape and then a pause of 3 seconds.

The tape-recorder was in the room with the patient. He was extremely nettled by it and relieved when its sound was drowned by that of the television in the patients' sitting-room on the other side of the door. He received 10 mg. of dexamphetamine sulphate with his 10 p.m. injection of apomorphine. When I visited him shortly afterwards his physical condition was excellent and I was not aware of any psychotic features in the patient. He had no injections at 2 a.m. and 4 a.m. and no further dexamphetamine that night. During the night he became acutely psychotic and frequently sought the nursing staff to tell them that the voices were telling him to carry out various actions—such as throwing his cup at the mirror.

When I first saw him in the morning, he eyed me very suspiciously. The following is an abridged version of a tape-recorded interview beginning at 10 a.m.

B. The first thing I remember was trying to disregard the voices completely. They seemed to be a series of disjointed statements about rubberized material. I did not see how it could be coming off the tape because it was such a small tape. I had a good look at it. I also had the feeling that there was a camera watching me because these voices seemed to know every move I was going to make before I made it, so that I listened and listened and gradually I cottoned on to what seemed to me a talk or a conversation between probably yourself and my mother. I thought—"Oh, Dr. Oswald must have gone to see my mother" and the way the conversation was going you were saying something about rubberized material. I think this was the voice of the Negro doctor who was in taking my pulse. Anyway, the next voice was a lady's voice. Not my mother's voice, but what I thought might have been my mother's voice saying something about rubberized material makes him cry or something. I am not quite sure about the wording, and then there was laughter. There was laughter all the time during all these conversations, but laughter followed this as if it was people laughing at an old wives' tale. First it started off as an unshaped word, but gradually it came to say "trousers", so that I thought and thought and thought and this repeated and repeated and repeated and I knew by this time that it couldn't be coming off the tape. This developed into various other things and I got up and spoke to the nurse. It was before my 12 o'clock injection. It was as if they had seen me getting up to go out of the door.

O. Who might have seen you?

B. The voices. I didn't hear your voice. I heard Dr. Fish's voice later on. He always spoke very quietly, as did the Negro. But these statements started again as if to distract my attention away from going out of the room. It didn't, of course. The nurse advised me just to come back, so I came back and sat down as if not to pay any attention. The disjointed statements started again, something about mackintoshes making me sticky, or sick. It was "sick" and "stick". I was trying to figure it out, because it is very true. When I have been wearing this to be sick it does stick to my body. The voices developed into trying to tell me that I was not really helping all I could; that they wanted me to be sick and they wanted me to do something about these waterproofs. I went out and asked the other nurse what I should do. I was bamboozled and she said she could not really help me. So I came back and I thought, well, they may want me to tear these things up, but that goes against the grain as far as I am concerned. They didn't say that, but they wanted me to do something about it. I threw my wife's one from this chair over to here, and I think I kicked this one that was up here. I just couldn't figure out what they wanted me to do at all.This cup was lying here and I looked at the cup. I picked up this cup and the voice kept saying, "There is nothing in the cup", and this recurred and recurred and recurred and it developed into these three staccato knocks which were a symbol of the cup, later on.

O. The clicks on the tape?

B. Yes. It came to be a symbol of me picking up the cup and smashing them. There was a smashing sound at first, and then the click took over as a symbol of the smashing and by this I took it to mean that they wanted me to pick up this cup and break it or throw it at the mirror which I told the nurse I might do, but then again I came back and said, "Why should I do it just because they tell me, because I don't want to waken up the whole ward and I don't want to break anybody's property." So things developed and it gradually worked on the theme that they wanted me to be sick. So I mixed this drink, I took a glass of diluted orange juice. That definitely was not enough. They were laughing at me. So I said, "Well, now I'll show the buggers", So I took the mixture and that still didn't make me feel sick inside and I sat at the basin here trying to be sick. This mackintosh came back and I thought, "Oh, well, they are saying I have got to be sick with this mackintosh on. I'll have to put it on." They said, "No, there is a mackintosh missing." I decided I wouldn't listen any more because I couldn't do any more. I didn't want to break anything. It must have been about ten past two.

O. Did you know it was a tape recording that was being played at this time?

B. I couldn't understand it. I thought there was something inside the box. I thought you were over in another building working it. I thought all the voices were there.

O. That I was influencing it from another building?

B. Yes.

O. You didn't think I was influencing your thinking?

B. No. I thought the whole group of the staff here were watching me somehow through this wee red eye in front which tells you when the machine is on. I dismissed it after a while. It couldn't be any telescopic lens remotely controlled or anything, but the voices went on to say something about this mixture, this dynamite, as if to say, "Really going to knock your head off when you are sick in the morning or when you are sick at all". Then I think there was a series of disjointed statements, then the statements started to take the form of sex relationships between man and woman, as being an ideal thing, of course. Each one was taking a part, about the stick, this stick I told you about.

O. Which is a penis to you?

B. To me it meant that, yes. It couldn't mean anything else. I think it was telling me that I should have normal relationships with my wife. I think I muttered under my breath once or twice when I was more annoyed. "What the hell do you expect?" "Why the hell do you think I am here?" So this conversation started to me to be just slightly obscene with the noises which were coming over. Grunting and—very quietly at first it came in. A woman's voice.

O. These were the noises of a couple having intercourse?

B. Yes. So it started my mind working along rather pleasant lines, and I started to drop off. The mackintosh started coming in and instead of me being the stick which the voices were saying, of course, the mackintosh was the stick. Then later on the mackintosh was called Brown Mackintosh and Brawn Mackintosh.

O. Your name, of course, is Brown?

B. Yes, and Jim. It was called Jim Mackintosh at a certain stage of the proceedings.

O. What was called Jim Mackintosh?

B. The stick.

O. You remember all this very well?

B. I was wide awake at this time, of course. I was sitting up the bed. I was mixing drinks, having a buffet. These disjointed statements started again. Mackintosh makes him sticky and the mackintosh makes him sick. They developed it to making a mackintosh out of a rubberized pillow case. This was Dr. Fish's voice. I felt that they wanted me to pick the pillow case up and throw it away. So I kept my own pillow just to go against them, you see. I think it developed and developed and developed and then the voices started to say that when I woke in the morning I would be violently sick with this dynamite drink—the stick, I think there's a connection between a dynamite stick and this carry-on with the mackintosh.

At 6 o'clock, when the nurse came in to take my temperature, she took away the cup and this voice was still saying, "There's nothing in the cup, there's nothing in the cup." She brought me a cup of tea by the way, in place of this other cup; and I kept looking at it and I thought, "That's silly. There's tea leaves in it." I piled the raincoats into the basin in order to be sick over them, but I couldn't do it.

O. The voices were telling you to do this, were they?

B. Well, I took it to mean that; I put one in, but they kept harping on this mackintosh business that you couldn't be sick without the mackintosh. The Sister came in and she seemed to me to be the symbol of the Gestapo; that this was a prison and I was in a madhouse and I started to worry. I was very puzzled. She watched for a minute and said, "You know you don't have to be here." * She gave me the letter; as I say one bit in it has a slightly nightmarish quality which is funny now I'm back in my normal senses. I read through it very quickly, and I came to this bit. "We had a most amusing episode this morning. Jean was up first, so she found your letter and gave it to me to read in bed. She went through, put on the fire and started to dress. When I was reading your letter again and again I noticed her going over to the chair, picking up something, walking through to the room about four times, then she told me that she had lost her brassière. I looked up and told her that she was havering and proceeded to read your letter. After she had walked back and forward about six times, she decided that if she couldn't find it she wouldn't wear it today."

Of course this sort of thing was nightmarish. It seemed to fit in with the whole ridiculous situation of me being in a prison. It just seemed she was saying, or somebody was trying to frame me for something.

O. Did you ever hear your own thoughts out loud?

B. I don't think so.

O. Did you feel that anybody was putting thoughts into your head?

B. I felt very strange when the voices always seemed to know what I was going to do next and they seemed to suggest my next action. Then I remember listening to these voices. It was slightly obscene and the sister didn't like the tone of it. She went out. I thought they were trying to make me want to do something I didn't want to do and what the Sister said seemed to fit in again with the nightmarish quality of the whole thing.

O. And when I came in and the tape was still going round and round, were you still hearing the voices?

B. Yes. Up to the time you bent down here and switched it off.

O. How do you feel about rubber mackintoshes?

B. I seem to have made a decision in my subconscious during the night to disregard them altogether. When these voices started to speak about mackintoshes I agreed with them that they made me feel sticky and unpleasant and they started to say that I should use this when they were speaking about the intercourse part about the mackintosh being a stick, and I didn't want this at all, you see, and I kept looking at the photograph of my wife, saying I wanted my wife and nothing else, and that is when I seemed to make the decision, in fact, I remember repeating her name over and over again to myself at the time when this was at its worst, so that I could say I was probably making a decision at the time.

He had bundled all the mackintoshes into a cupboard. No 10 a.m. injection was given. At first he refused his 12 noon injection and refused to look at the mackintoshes, but was persuaded to continue. The tape-loop was re-started at 5 p.m. No amphetamine was given that evening. By 10 p.m. he was hearing the voices again saying things quite different from those on the tape. He was unwilling to talk about them and eyed me suspiciously, but spoke of "a mixing-stick" and "mix it thick" and "stir it thick", which he took to refer to curdled milk in his stomach, together with more sexual intercourse noises. He was aware of a witch stirring his abdomen with a mixing-stick (cp. "makes him sick"). The wooden stand around which the tape-loop was passing was swaying because the loop pulled slightly on it and he felt this had a special purpose. He said the voices were making up rhymes. The tape-loop was stopped at midnight and he had no further injections, except one last one of apomorphine at 10 a.m. the next day.

* The Sister subsequently denied this.

Two days later, though otherwise apparently quite rational, he still was scarcely convinced that there were only the few repeated phrases on the loop. He had worked out that they were on half of it only and that the other half was being varied by people watching him. He believed that there were definite long silences. The voice saying, "There's nothing in the cup" was "a clue" to the whole thing. He was baffled by the fact that at times the word "sick" was endlessly repeated, but then changed to "stick". He wrote the following description of an autoscopy experience he had during the first night of voices:

"I was lying in bed listening to the voices. They came from the corner of the room where the tape-recorder was playing. In the state of mind in which I found myself, I seemed to visualize, I think, three figures in the corner of the room where the recorder was. Two of the figures were standing, one with its back to me, and one vaguely in the background. I seemed to be sitting with my head in my hands, trying to be sick. I knew it was me, but I couldn't see my face. The woman's voice which I had been hearing all night was speaking. The voice was trying to make me sick. I don't recall the words used, but I remember that they were said in a teasing tone, as if I was a child being coaxed to do something. I replied by lurching forward over the basin which was my constant companion all week, and making imbecilic noises whilst trying to be sick. Of course the usual laughter followed.

"At this time I was lying in the room, awake, on the bed, but I thought that it was not the same room in which I had been during the night. It seemed to me that it was a similar room, with the same furniture, but in a different place.

"I was certain I was being used by some maniac called Dr. Oswald. I was certain that I was being made into some robot, or something similar, to carry out the will and wishes of the said maniac."

He experienced derealization and depersonalization during the following week while he was out of hospital, but this soon vanished.

After 21 months he remains well. He feels quite indifferent to rubberized clothes and finds it hard to believe how he could ever have had his interest in them. His career has prospered extremely well by his own efforts and talents, and his wife confirms that they are normal and happy in their general and sexual life. She has had a baby and, though he masturbated during the end of her pregnancy, he denies any interest in fantasied or actual mackintoshes on those occasions. No other deviations or nervous symptoms developed and he is really a very normal young man. He has had no further experiences of a schizophrenic nature and it is true to say that we are on excellent terms with one another, the suspicious animosity present at the end of the period of aversion therapy having quickly vanished.

Case 2. A 38-year old man who had been working as a miner and as a bookmaker's clerk for four years, having previously been a ship's cook. His first marriage ended in divorce and his second was foundering because of his addiction to alcohol. He had drunk mainly beer and whisky increasingly over ten years, had once been auditorily hallucinated after a heavy rum-drinking bout several years before and now spent most of his income on drinking round the clock till his money had gone. His wife had refused to sleep with him for several months owing to his drunken state and he had reverted to an earlier fetishistic practice (he stole women's underclothing from washing lines, masturbated into it: and then threw the clothing back).

He was treated with apomorphine, pilocarpine and dexamphetamine like Case 1, and after each apomorphine injection drank whisky and beer.

On the third night a tape-loop was played:

"Drinking beer makes him sick." Vomit noise. Laughter. "A father! A husband! Beer and whisky make him sick." Vomit noise. Laughter.

It was particularly striking in his case how he tended to retch and vomit at a rhythm determined by the vomiting noises on the tape. He did not experience changes in the voices.

He did fairly well and after 20 months his wife is still satisfied with their marriage. He drank beer again for the first time when his mother died seven months after his treatment and now drinks about once a month. He still cannot face whisky.

Case 3. A 32-year old N.C.O. in one of the Services. His chief delight was to be tied up or tightly enclosed in black, shiny rubber. He recalled that as a small child his mother had forced him to wear a black rubber mackintosh like his sibs wore, against his will. He insisted that his fetishism began when he was 17 when, after one week in the services, some other youths seized him, tied a groundsheet over his head and masturbated him. Since then he had made a practice of tying himself up with rubber groundsheets, a rubber hood and ropes. He came for treatment partly because he feared he might encompass his own death, as he had recently had difficulty in releasing himself. There was no family history of mental disorder.

He had had a couple of mild flirtations with girls in his life and had once had sexual intercourse at a London brothel specially equipped with rubber strait-jackets, hoods, thongs, rubber zip suits and so on.

He was treated on similar lines to Case 1. After each apomorphine injection he tied himself up in groundsheets or donned a black frogman's suit. He had 10 grains of dexamphetamine sulphate each of the first two nights. On the third day vomiting was decreasing and he received 1/10 gr. pilocarpine nitrate as an extra. In the evening the following tape loop was started.

"Rubberized clothing makes him sick" (male voice). Vomiting noise. Female laughter. "Tying up with rubber, it's ridiculous" (male voice). Vomiting noise. Male and female laughter. Pause for 5 seconds.

He received 10 mg. of dexamphetamine sulphate at 10 p.m. His extreme dislike of the tape-loop was very plain and he switched it off himself in the night and could only recall a little of what he had experienced. The following is an abridged version of a recorded interview at 9 a.m. the following morning:

O. Tell me what you were saying about how the voices changed in the night.

P. Well, they were to do with electrical impulses or something. I seem to remember reading about it.

O. You told me you heard something.

P. "I tied up my brother."

O. Did it mean you?

P. No, I don't think so. I think it was the person who was saying it. That's the impression I got. They kept saying it for a while, and I thought it was my hearing. And, "My clothing made me sick."

O. It didn't say, "Your"?

P. No. Getting towards this morning there were a lot of jumbling voices. I couldn't really understand it. But I told the sister there must be something wrong with the set. I didn't think it should make all these noises. They were talking. I can't remember what they were saying.

O. Were you puzzled by this?

P. Yes, I was. We switched it off. I thought it might be the heat or something like that.

O. How did you think this was being brought about?—this change in what was being said?

P. I thought I had read something sometime or other that a tape recorder does do that. It skips a word or it skips a meaning or something, and you get the totally opposite meaning to what you should have. Like, I say something to you and after a while this grows into something else. I never went to sleep. There was one other. Something about "smother", I can't remember. It was just making a remark.

On the evening of the same (fourth) day he walked out of the hospital and travelled 80 miles to his unit, but could not sleep or eat, and at 4 a.m. he walked 5 miles to a station and got back to the hospital at about 8 a.m. The treatment was then resumed. He aggressively rejected the rubber garments and only donned them on firm persuasion. The tape-loop was restarted in the evening, but was later found not working. Examination showed that he must have unscrewed the electric plug and disconnected the live wire. He had no amphetamine that night (it was 48 hours since the last dose). Once again the voices changed and he switched the tape off at 4 a.m. At 9 a.m. the following was recorded:

O. You switched the tape recorder off at 4 o'clock. Why was that?

P. I was hearing things different from what there was there.

O. Do you mean that the things you were hearing were put on to the tape during the night?

P. No.

O. How did they get there then?

P. They must have been—
different meanings and sayings. It said one thing, and you got it wrongly, you know . . . example . . . "Zonial clothing goes in the sea." Other things . . . I was going to write them down. I wish I had.

On two subsequent days he received a number of massive doses of testosterone by injection and was given books of "art" photographs of the female form to study. He felt enormously energetic, but was not greatly interested in the photographs.

He tried out his rubber articles a week after leaving hospital, found they held no interest for him and discarded them. He went out to dances and other social events for the first time in years. After 6 months he relapsed and a further 4 months later made known his deviation to the Service authorities and was invalided. He told me at that time that he intended to live in London where there were others who shared his interests. He had been back to the brothel which, he pointed out, advertises in a well-known week-end publication available at any bookstall, under the guise of a rubber-clothing store. He had formed a friendship with a male homosexual (but had not had sexual relations with him), whom he had first noticed wearing a black, shiny, rubber mackintosh in Hyde Park one fine summer's evening.

Case 4. A 31-year-old alcohol addict. He had started drinking in the Navy which he had joined a few weeks after his marriage, when aged 21, in order "to avoid Korea" (he had first been called up for the Army). He had often reproached himself for this cowardice. The marriage was not consummated for three years and relations with his wife were often difficult. After

leaving the Navy he drank more and more heavily, mainly beer and rum. He was in trouble at work, for he would drink several pints at lunch, leave work for more between 2 and 3 p.m., and then would spend the evening drinking. His surname we shall call Jick (it rhymed with sick).

He received two-hourly apomorphine injections before drinks of beer, Guinness and rum and "coke", with 10 mg. of dexamphetamine at 2 a.m. On the third day a tape-loop was played as follows:

"Beer and Guinness and rum and coke make him sick" (female voice). Vomit noise. Female laughter. "Drink, Why?" (female voice). "Is it all because of her?" (male voice). Vomit noise. Female, then male laughter. "Makes him sick" (female). Click and three seconds pause.

He was very nettled by the tape and late that evening refused his injection and unplugged the tape machine. However, he was persuaded to continue.

The following morning he reported that what the tape had said had changed in the night and that it had, in fact, happened before he unplugged the tape on the previous evening.

O. Were the voices saying the same thing all the time?

J. No, no. Sometimes the actual voice changed. I can't remember what they were, I wasn't paying attention. Sometimes it was like a party that was going on. The next minute when you stopped and listened it wasn't. I was even noticing it this morning before you came in. I was not very sure of some of the things they said, they seemed that ridiculous.

He described a sort of dream about what he called an "auto-psychoanalyser". "It was a machine like a tape-recorder, just a loop like you've got there. You fed your troubles in and it would all come out back again with all your troubles put right. It was that real I thought it worked on people."

On the fourth day he received a booster of 1/10 grain of pilocarpine nitrate. That night he did not receive amphetamine. The next morning he said the tape-recorder had been changing again:

J. It changed and I couldn't get it back to its own words again. I couldn't remember what the proper word was. "Was it all because of her", or something, it changed—a Service thing—"shot of", "get shot of" or something. The first bit, too, it was "Fear and Guinness".

O. Did you take this as something to do with you?

J. Yes (emphatically). I reckon I've always been afraid in my life. This room seemed to be out of this building, on its own somewhere.

The aversion routine was stopped at 9 p.m., until 9 a.m. the next day and then from 5 till 9 p.m. the tape loop was played.

At 9 p.m. he said he had kept hearing "Miss Jick", or his wife, "Mrs. Jick" mentioned and evidently this had been instead of "makes him sick".

After 11 months he still drinks no alcohol and he and his wife say they are happier than ever before.

Case 5. A homosexual male aged 25 under the management of Dr. W. D. Boyd. The patient's tyrannical father died when the patient was 15. The mother, to whom the patient was closely attached, was a drunkard, a spiritualist and a Lesbian. Elder brother had never engaged in honest work. No family history of mental illness.

Homosexual relationships began at the age of 14 and included frequent brief affairs in public lavatories and several long love-affairs. He had married at 19 and had two children. He came for treatment to try and salve his marriage, for he had been living away with a man for eight months. He was usually a passive partner, principally interested in fellatio. He was greatly excited by male urine and sometimes drank it.

Aversion therapy was embarked on with some reservations. A 30-minute interview in which he described his homosexual practices was tape-recorded. Every two hours he received apomorphine by injection and then the tape-recorded interview was played through a loud-speaker. Glasses of urine were sometimes placed by him. In the intervals the following tape-loop was played; "It makes him sick, it makes him sick. Sex with men? Oh, it makes him sick now. He gets sex with men. It must make him sick now. He'd meet men in the lavatories. Ugh. Sex with men makes him sick. He looks at men's bodies. It must make him sick now." (One male voice). Four seconds pause.

He received pilocarpine nitrate 1/20 grain once on the first day and once on the fourth, and dexamphetamine sulphate 10 mg. each night. Fluid and electrolyte depletion were prevented as with the other patients and serum potassium and sodium remained within normal limits, though the CO_2 combining power rose to 33·5 m.Eq./L. and chloride fell to 96 m.Eq./L. on the fourth day, having remained within normal limits previously.

He experienced the words of the tape-loop changing from the first day and throughout the four days' treatment. The changes never had any great significance for him, seeming merely trivial or absurd. He could not voluntarily re-experience what was actually played through the loudspeaker. The changes were experienced by day and night throughout the four days and three nights of treatment.

At times he wrote down the various phrases he heard, which included:

"I like it thick my bacon thick. Sanford man. what makes it him sick now. He has Sangford man. They musta made a mistake now. They'll need 8 men in the lavatories. Sangford man makes it sick. Be a good man Sportis? Do not make sick now. I'll knock him sick. What makes him sick now. Enough with men's bodies. Bolton quick it'd make you sick. Sex written in. He's got six sick men, that must make him sick now. He'd eight men in the lavatories. Sax written back Matron's sick. Hey'n they've got nice bodies. But you mustn't make them sick now."

After the end of treatment on the fourth day he wrote of his most recent auditory experiences:

"This impression of the tape-recorded message was not written down in detail at the time I heard it because I was quite convinced that it really was a separate recording, and I accepted it as such without question. I remember that the dialogue seemed much briefer and with longer pauses than the original recording, also the intonation seemed different. While listening, my mind's eye formed a picture of the characters involved in this little 'sketch'. One person monopolized all speech on a telephone while his weak-stomached friend sat immobile in a bath-chair. The man on the 'phone would jokingly say he had 'mixed bodies' (sweets?) then say something quietly to the effect that he had better not say that as it might make 'him' (the bath-chair sitter) sick. There would then be a deep belching sound not before heard on the recorder and the person on the 'phone would say, 'He's sick now,' or words to that effect. Then after a pause, very loudly, 'Oh, we've made 'im sick, we've made him sick,' then carry on more normally asking after the health of two friends with strange names I cannot remember. The dialogue would then come round to the part about 'mixed bodies' and carry on repeating endlessly the same sickly tale. If I remember correctly these variations in the recordings always came after I'd snatched a little sleep. They never changed while listening and no effort would make them sound like the original recording."

A month later the patient reported that he had had one attempt at sexual relations with a man but had stopped because he felt feelings of revulsion and physically sick. Within two months, however, he had left his wife and gone off to live with a man.

Case 6. A 37-year-old transvestist, a telephone engineer, who had as a child obtained sexual satisfaction from donning his sister's knickers. As he grew older his dressing-up became more and more elaborate. When aged 21 a surgeon had advised him to wear elastic stockings all the time. On occasion he would wear a stuffed brassière, skirt and blouse, cover his short hair with a scarf and apply lipstick and mascara. He had married at the age of 23 a woman who had had previous sexual experience. There were two children, a girl aged 13 and boy of 11. The latter had spent much of his life under a child psychiatrist and was, at the time, at a special school; he had an I.Q. of 140, but among other troubles, persistently and apparently deliberately defaecated in small quantities into his trousers.

The patient had a strict Nonconformist up-bringing and conscience, and experienced much guilt from his practices. He had never given full sexual satisfaction to his wife because of his "brotherly" attitude to her. She had been out at work and at the time of his admission she was conducting an affair with a man she had met, though the patient was not aware of this. Her affection for the patient remained considerable. Until the time of his admission she had avoided questioning him in detail about his dressing-up practices although she knew of them. When he confided more details to her, she threatened to leave him unless he was cured. He expressed great fear lest the desire to walk along the street in female garb, which lately had been becoming overwhelming, led to his arrest. He had been going as far as putting out the milk bottles in female clothes a couple of times a week. Although he did not know of his wife's extra-marital adventure, it is probable that a change in her attitude led to him seeking treatment.

He gave a history of fantasies of spankings when dressed in female garb, and occasionally when so dressed he indulged in self-flagellation. He fantasied being dressed up in order to achieve orgasm with his wife, but found her sexually exciting in her own right at times. He also reported wet dreams in which normal heterosexual fantasies were present.

The brief outline of his case given here will indicate that he was never regarded as an ideal case for aversion therapy, but, partly owing to the success with Case 1 of this series and because Case 2 had not yet relapsed, it was decided to try such treatment with this patient.

There was no family history of mental disorder requiring hospital care, but the patient's mother had hysterical "collapses". She often told the patient when a boy that she had wanted a girl. He had been much more attached to his father, a quiet, gentle Civil Servant.

A single room was hung with mirrors and a large variety of female clothes (which at first obviously thrilled him). He received 2-hourly injections of apomorphine 1/10 grain, after each of which he dressed up in various of the clothes, applied lipstick and was sick. No amphetamine was given. Two injections were missed on the second day, three on the fourth day, five on the fifth day and on the final day he received only four injections. On the fourth day pilocarpine nitrate 1/10 grain was twice added, as was the case on the fifth day. On the other five occasions on the fifth day and on all four final occasions pilocarpine nitrate 1/50 grain was added to the apomorphine. Clinically he did not appear dehydrated; by the fifth day his serum potassium had fallen to 3·8 m.Eq./L. and CO_2 combining power had risen to 33 m.Eq./L.

On the fourth day a tape-loop played through a loudspeaker was started. It went as follows:

"Dressing-up in female clothing makes him sick" (male voice). Vomit noise. Laughter (male and female). "Him! Trying to be a woman! It's ridiculous!" (female voice). Vomit noise. Laughter. "Wearing brassières makes him——" (male voice). Vomit noise. Four seconds pause.

From the first he tended to vomit after his injections at the time of each vomiting noise. In the evening he asked after the female voice, "She's nice, she seems to be on my side." The following morning he said that the voices had been changing:

T. It changed a lot. All except the woman's voice. Her voice never changed till about 5 o'clock. I can't remember. The male voices—something about "Let him get dressed with the infant females." It said that for quite a long time. The other fellow, I can never make up my mind whether he was saying, "When he wears a brassière he blushes" or "he cries." I don't even know now. It changed so completely. Then the girl said something very ridiculous this morning. I was quite disappointed, because she'd been going in good style all night. If I'd known you'd want to know, I'd have made a note of them. It would have given me something to do.

O. The woman's voice changing—was it unpleasant, about you?

T. I don't think it was about me. The beginning part of it changed, "ridiculous" stayed clear. It was just trivial. If there had been anything that I'd taken personally I would have remembered it.

O. What the men said, this was personal?

T. This was personal, yes. I didn't sleep enough to dream, I can be quite definite about that. The two men were rather sneering, ending up with "cry" or "blush" or "sickness" or something like that. Later she changed. The others changed completely right through. Sometimes it was gibberish, but other times it was just a twist on what they were saying, you know. The first voice could never make up its mind whether to say the same thing twice or not. The other voice I concentrated on because I was trying to make out why he was saying "cry" or "blush".

O. How do you think this happened?

T. I thought you had a continuous tape on, that it was the tape varying, to be honest. It came clear again this morning, so it must have been my hearing.

O. When I came in it was clear? What was it saying?

T. Um. (long pause). Ha, I was listening all night, I should know. Um. (10 seconds pause) Oh dear! I should know. The first one says, "He puts on female clothing, makes him sick." The girl was saying, "Him. He wants to be a woman? Ridiculous!"—as if she doesn't believe it, you see [in fact, her tone was one of scorn that he should try]. The other voice was saying, "When he puts on brassières he has to cry."

The tape machine was switched off at 9.30 a.m. At 2.30 p.m. a new tape was begun, in which all the speech and laughter was by females (three) and the vomiting noises by a male. There was a 15 seconds pause in which were a few noises which, admittedly, could be thought to sound like the growl of a lion—the noises arose from a male voice previously recorded at a faster tape speed. The tape ran as follows:

" Brassières, panties and skirts make him sick." Vomit noise. Laughter. "Huh! Him! He could *never* be like us. Trying to look like a woman makes him sick." Vomit noise. Laughter. "Undies, stockings and lipstick make him sick." Vomit noise. Laughter. Pause with three clicks and a few roaring noises.

On the last and final morning at 9 a.m. the tape machine was switched off. It transpired that he had been crying and had refused one injection in the night. He said everyone was against him. He had written down some of the things he had heard. At one time he had heard me ask him a question, but when he looked I had gone.

He had noted that at midnight there were conversations about, "Making different women out of chocolate," "stirring it thick," and making other things out of chocolate.

He made a series of written notes between 2.45 and 3.15 a.m., which he explained to me. It was at this time that conversations with a lion were in progress.

T. There must have been a lion, yes. The lion was roaring in the background. Everything was said to the lion. I looked at my watch when I first noticed it was going away from being what it was. I heard, "It could never be like us. Your highness looks like a woman would make him sick." First of all it was, "though a woman would make him sick." They were addressing the lion. Then preceding that, "Laughing at cafés and carrots and Nathean ticks." Then at 3.10 it came round to this version, "Laughing at Country and Kirk and Napethian tick. (Roar) (Laughter). Oh, he could never be like us. Trying to look like nothing would make him sick. (Anguished roar, laughter)."

O. Was this you they were talking about?

T. No, they were talking about the lion, he was roaring back. "Only doggerel and lipstick make him sick." That's the only time the lion actually says anything you can hear, it was a roar of, "Doggerel". He was definitely indignant. He was hurt that they should regard doggerel as making him sick. At 3.15 the line, "Your Highness . . ." was reintroduced. There were variations in that last line. I remember once "Bumblebees and doggerel and lipstick," and other times it came over as "Carrots doggerel and lipstick." Then it went to this very stupid version. It kept it up. I didn't write that down out of one go. I got a line down each time. It was so quite definite I even had time to correct myself.

It sounded rather like a modern poet. The sort of jargon nobody understands. At 5 a.m. it was, "Lavian, cafés, and scraps of Nathian thick (Growls, laughter)." "Her and him could never agree and I can't try to look like a woman licks him, slick. (Sorrowful growl, laughter). Undies, doggerel and lipstick make him sick." Again the lion was objecting to the doggerel.

The laughter was very pleasant, just like children, entirely different to what the laughter was in actual fact. That was definitely nasty, but not this laughter, it was changed round in character completely. It was children's laughter, not adult laughter.

O. Do you really think a lion could talk to ladies?

T. (Pause). No, I suppose not. You hear things like Lennie the Lion and it could just as easily have been a puppet couldn't it?

He could not recall the original version of the tape. At one time he had left his room in the night and sat in the sitting-room, but it seemed to be filled with the smell of ether, so he went to the bathroom, "It was nice and peaceful there."

At 4 p.m. the same day the tape-loop was re-started. At 10 p.m. he asked me when it was that I had changed the tape again and put on children's laughter with different words. He seemed detached and quiet and was fully oriented, talking normally. He sat and listened over and over to the tape-loop and wrote down, bit by bit, what he heard, correcting it as necessary:

"La bien. Panties and skirts and mink skins. Oh, in the end could never be like us.

Try to look like a woman { dressed / mink skin pink

Unaided lipstick make him sick."

He was quite positive that this was a new tape and that it was not his own mishearing. The lion was roaring, and children laughing at the remarks, which were not related to himself.

The tape machine was stopped and 6 grains of sodium amytal and 50 mg. chlorpromazine given. He slept till 9.30 a.m. the next morning. He was then still sure the remarks he had heard the previous evening were genuine. In the next week he tried to "date" an attractive, young female nurse who worked in the ward. A further brief course of apomorphine with dressing-up, supervised by that nurse was instituted. Whereas he had always previously been childish and dependent, he now showed considerable aggression. He had wanted to appear a "he-man" to this nurse and hated and rejected the female clothes. In the following weeks he was agitated, he threatened suicide and wept frequently. He gradually improved and returned to work after ten weeks, having had a flirtation meanwhile with one of the female patients. In the 8 months since, he has dressed-up in female clothes on many occasions, but notices a striking change in his fantasy-life, the dressing-up fantasies and masochistic fantasies that he previously indulged in daily having disappeared.

Case 7. A 22-year-old man. Father a heavy drinker, divorced by mother when patient aged 9. She remarried and had two more children. Patient was always a behaviour problem as a child. Went to sea aged 16 till 19, but in constant trouble owing to bad-temper and excessive drinking. Since then innumerable brief jobs lost because of rows when drunk. Several suicidal attempts, the last two serious. Mainly gin and beer: gave up breakfast three years before. Admitted for treatment of alcoholic addiction and possible rehabilitation. He had an air of self-confident, cheerful affability.

The effect of a tape-loop alone *without* apomorphine or other drugs was first investigated. He was confined to a single room and wakened hourly as a patient receiving aversion treatment would be, and he held a gin bottle in front of him for 5 minutes each time. The following tape-loop of 40 seconds duration was played through a loudspeaker:

"Wreckage" (male voice). Laughter. "Gin and whisky rotting his guts. Rotting his mind" (male voice). Laughter. "What he could be instead. Hmm!" (female voice). "I can see his brain and body rotting. Listen!" (male voice). Noise of liquid being poured into glass. Laughter, 10 seconds pause.

On the second morning he reported that the tape-loop had been changing. He wrote these things down and listened carefully to them in my presence. He heard one change while I was present, "There! You heard that, didn't you!" During that afternoon he continued to hear changes and had no doubt they were genuine and thought someone was arranging it in another room.

The changes involved the second half of the passage and included the following heard remarks:

"Oh, what could Ian said?" and "Oh, what did Ian say?" (obviously derived from, "What he could be instead").

He remarked that he did not know who Ian was, but whoever he was, he replied to the female questioner by saying, "I said this is Raymond's body rotting" or "Raymond's brain is going bloody rotten" or "Oh, Raymond's mind is going bloody rotten", or "I can see his brain is bloody rotten." He had no idea who Raymond was.

The third night he threw a chair at the loudspeaker high up on the wall and said he was "hearing things" and, "I must be going out of my mind." The proceedings were terminated. Two nights later he got drunk.

He subsequently received a course of apomorphine aversion treatment and on the third day a new tape-loop was started. He was furious with it, and when given salty soup to drink at the odd hour, retched repeatedly in rhythm with the vomit noises of the tape. He walked out of the hospital unobserved that evening, dressed only in a boiler suit and plimsolls he had hidden. He slept on park benches for three nights with no food, and then returned and asked to start again. A week later he was started on a new course of aversion therapy lasting four days with 10 mg. of dexamphetamine sulphate each night and pilocarpine "boosters". The tape, loop used the previous week was re-started. He spent most of his time, like other such patients do, with his head under the blankets so that he could not see the bottles and bowls of vomitus round the room, nor his own reflection in a large mirror. In addition, he held his hands over his ears. He was less co-operative in saying what he heard, but claimed that the only change was in a phrase "Drinking, drinking, drinking, etc." which changed to, "Ding dong, ding dong, etc."

He behaved in a pleasant and likeable manner in the next four weeks, visiting a public house with friends once and drinking lemonade. He found their jeers hard to bear. He was sent to an Industrial Rehabilitation Unit, but resented the discipline and seeming triviality of the tasks, absented himself, drank beer and was sick. Again the next day he drank and was sick, but after that he drank heavily for several days. After 4 months he drinks beer a couple of times a week, but no spirits.

Normal Volunteers

Studies with normal volunteers are under way, but it may be worth noting here that the phenomenon of changing voices, with tape-loops comparable to those used with the patients, is found with normal subjects who are not sleep-deprived and who have received no drugs when they are subjected to a few hours relative "sensory deprivation" (apart from the auditory stimuli).

Discussion

The Voices

Titchener (1908) described experiments which indicated that our perception of the order of momentary, almost simultaneous events can easily be at fault. In 1958 when interested in devising techniques for the investigation of discrimination during sleep (Oswald *et al.*, 1960), we made loops of tape into which were jointed three adjacent short lengths on which tones of differing frequency had been recorded, so that a tone-pattern of total duration $\frac{1}{3}$ second (comparable in length to a word) was played repeatedly at, for instance, 12-second intervals. Although we did not find that an occasional tone pattern in which the order of the three tones was changed was sufficient to cause awakening from sleep, we were intrigued because we found ourselves unable reliably to perceive the correct order of frequencies within the repeated pattern. We were therefore interested in a brief note by Warren and Gregory (1958) announcing the discovery of an auditory analogue of a reversible visual figure. They reported that when the word "rest" was repeatedly played without any pauses, normal subjects tended to hear it change to *tress* or *stress* or to *Esther*. The subject misperceived the correct order of the phonemes in the word and even perceived an extra one. Their work was not borne in mind, but subsequent to the rather unsystematic and exploratory observations reported here Warren (1961) published an account of further studies which are highly relevant to the present work.

Warren cut single words, or groups of words up to a total of four syllables, from tapes and made these into loops containing no pauses. The repetition rate was thus extremely high and subjects heard the loops for three minutes only. Despite the discrepancy of techniques, Warren's observations are similar to those described here. Warren also found that the greater the number of repetitions in the three minutes and the greater the clarity of speech, the more it was heard to change. As in the present study, his subjects all thought that what they were listening to was genuinely changing. Again, while most of the changes were understandable mishearings, there is evidence in his studies too of florid misperceptions. When *Rape, rape . . .* was repeated 435 times in three minutes, sometimes *pray*, but also specifically *prey* was heard; in one case changes included *rake, break, wrench, wench, drench* and *quench*. Warren stated that his subjects "seem to feel that they are not revealing anything about themselves by their responses, but simply reporting the sometimes foolish things the voice is saying".

A group of workers at Montreal has reported the therapeutic use of repetitive "verbal signals" suggesting that the patient is, for instance, liked by others and of a warm, friendly and affectionate disposition. These have been played for weeks at a stretch. It appears that no great interest has attached to possible misperception, but Cameron *et al.* (1959) noted that "under certain circumstances" the patient "may alter the meaning of the signals" and that, for instance, "a patient on being asked to repeat a signal to the effect that he was getting warmer and more friendly might say, 'The voice is talking about the present monetary crisis in Japan.' "

In the present studies the words spoken and the manner in which they were spoken, as well as the laughter, were designed deliberately to disturb the patients emotionally. The tape-loops used were intended to introduce a "social" element into the treatment situation and a consequent paranoid attitude might have been expected. The original rationale was that by such means a maximal emotional crisis might be attained, and subsequent "conversion" thereby facilitated, as Sargant (1957) would propose. The view that the strength of learning depends only on the number of repetitions was not accepted, the belief held being that the strength of learning also depends upon the degree of personal significance of the stimulus situation and on the degree of attention, or "arousal", so provoked. It was hoped that the words would recurrently force the patient to think distressing thoughts about human relationships and practices, instead of retreating into an inert, if disgusting, condition. The tape-loops certainly helped to make some patients retch and vomit. Cases 1 and 4 both seemed to go through a crisis in which the tape-loop played a considerable part and the therapeutic result was good in each case.

Cases 4 and 7 reported hearing the voices say things that can be understood as mishearings. Cases 3 and 6 reported less understandable remarks, while Case 1 reported remarks and feelings comparable to the experiences of acute paranoid schizophrenia.

Granted that there may be individual differences in response, the question naturally arises whether these techniques could be modified to provoke brief episodes comparable to acute paranoid schizophrenia in a proportion of volunteers. It might be that if subjects could be induced to carry out repeated actions about which voices spoke in words based on their own introspections, perhaps mixed with words of their own, this could be done.

Hallucinated voices are common, though quickly forgotten, in normal persons during brief episodes of drowsiness. These hypnagogic hallucinations

are accompanied by other schizophrenic-like features of thinking during drowsiness (Oswald, 1962), and since the drowsy person continually flits between a state of lowered cerebral vigilance, or light sleep, and a state of wakefulness, the hypnagogic hallucinations and other phenomena seem subjectively to intrude while he is awake. Stimuli recurring repetitively at intervals of half-a-minute or more, or even at intervals of a second or so, provoke repetitively the EEG changes of brief, lowered cerebral vigilance or light sleep (Oswald, 1959a, 1960). At least some of the more absurd things that some of the patients reported having heard could therefore be tentatively classed as hypnagogic hallucinations. Since sensory deprivation provokes drowsiness and hypnagogic hallucinations, the experiences of the normal subjects mentioned are not incompatible with such a view.

The occurrence of schizophrenic-like experiences in sleep-deprived persons has been reviewed (Oswald, 1962) and can again be ascribed to the fact that they undergo repeated momentary episodes of light sleep. The patients reported here were all somewhat sleep-deprived, for they were repeatedly woken at night, getting only short naps by day and night. This, too, would be in favour of ascribing their misperceptions to drowsiness.

What, however, of those instances where, apparently wide-awake, they listened and wrote down what they wrongly heard in my presence? And what of the subjects of Warren (1961)? Warren has pointed out difficulties in the way of the original view (Warren and Gregory, 1958) that the experiences were analogous to those with a reversible visual figure. Certain modern psychologists have written of the accumulation of "reactive inhibition" or "conditioned inhibition" when a stimulus repeatedly evokes a response. These are psychological terms which, it must be said, are too often used as if they had some established physiological correlates. They are derived from Pavlov. Pavlov believed in "localized" or "partial" sleep or local cortical "inhibition". In fact, there is some electrophysiological support for Pavlov's belief that cerebral vigilance can fall in functionally-localized groups of neurones (Oswald, 1962), and though we could not ascribe all the auditory misperceptions described above to generalized sleep (which Pavlov identified with generalized cortical "inhibition"), we might be allowed to suggest that it is related to fall of cerebral vigilance in those functionally-localized groups of neurones concerned with perception of speech, or that "conditioned inhibition" is responsible.

Some of the patients had dexamphetamine, but others who did not receive that drug had comparable experiences. Case 1, whose experiences were most dramatic, on the second evening when he heard the voices saying strange things, started to do so and ceased doing so when the tape-recorder was switched on and off respectively, nearly 24 hours after his last dexamphetamine. The doses he had earlier received were, in total, trivial compared with those which, in total, precede the development of schizophrenic-like psychoses in dexamphetamine addicts. It may be noted that while Cases 1 and 3, both fetishists, heard hallucinatory voices, Case 2, also a fetishist, did not, yet he was the only patient to have been auditorily hallucinated before in his waking life.

The American psychologist B. F. Skinner (1936) made recordings of patterns made up of spoken vowel sounds and played these very quietly to subjects required to say what words they thought they heard. Each pattern was repeated a dozen or so times. Subjects tended to hear nonsense phrases of definite words. On hearing the same pattern again they would often report a different phrase in which, however, the same word might occur, yet having a different meaning and context. The play on the word "stick" by Case 1 is comparable.

Skinner suggested that the type of misperception shown by his subjects was comparable to that of the paranoid patient who overhears criticisms of himself.

"Behaviour Therapy": Some Clinical Considerations

Raymond (1956) first reported the successful treatment of a fetishist by a course of aversion treatment. I understand from Dr. Raymond (personal communication) that his patient relapsed, though not to his former, severely affected state, and received a further course of treatment in 1960, since which he has continued to eschew perambulators and handbags. Two fetishists described here were rubber-clothing fetishists, one of the most common varieties of fetishism. Psychoanalytic explanations of fetishism usually quote cases of shoe or fur fetishism with their obvious genital symbolization. How can one explain the appeal of rubber clothes? One of the three cases of fatal plastic-bag fetishism described by Johnstone *et al.* (1960) was also wearing rubber garments. One wonders whether in the use of rubber (or today, plastic) baby-pants one might seek for an origin.

Barker *et al.* (1961) and Glynn and Harper (1961) have each described a transvestist treated by aversion therapy, who, after 3 and 7 month follow-up respectively, at the time of their reports had not shown indications of relapse. Their patients were aged 22 and 27 respectively. Case 1, a fetishist, described here, was aged 22 and seems cured. Case 3, who relapsed, was aged 32 and the transvestist, Case 6, who relapsed, was 37. Raymond's case, who also has shown relapse, was aged 33. Had Case 6 been treated when he was only 22, one would have had to deal with much less complex dressing-up practices, entrenched much less firmly by the years. One must respect the importance of age in determining success in treatment. Case 6 is an excellent illustration of the fact that aversion therapy can be followed by a period of worsened psychological disturbance, when traditional treatments based on personal relationships or "the transference" and the passage of time are, I believe, essential.

Aversion treatment has now been accorded an honourable place among the techniques of "behaviour therapy" (Eysenck, 1960). Behaviour therapy, Eysenck states, has no need of the adjunct of the personal relationship, which may fortunately be relinquished so that relatively unqualified assistants may conduct the time-consuming parts of the therapy (Eysenck, 1960, p. 19). Such a conclusion could not, I believe, be reached by anyone who has had experience of immediate responsibility of treating such patients. The *success of aversion therapy is vitally dependent upon personal relationships.* The therapist must establish a sufficiently good relationship with the patient first to persuade the patient to start upon what, he must be warned, may be the most terrible experience of his life, and secondly, to sustain the patient throughout the treatment when, as invariably happens, the patient angrily rejects the whole situation and demands his clothes and shoes. If such a relationship is established, the patient may walk out but will return, as did Cases 3 and 7.

The therapist must visit constantly by day and late at night. During the period in which Cases 1–7 were treated, I saw two other transvestists, one exhibitionist and several alcohol addicts whom I would have treated similarly, but who would not come into hospital because there was not a strong enough bond between us. One alcoholic was under treatment in a week when my car was out of order; I could not visit him constantly, he walked out on the third day, and though I saw him later he would not restart.

The banner of behaviour therapy will not be advanced if claims are made for it by those who reject what experienced clinicians know to be of value. It

can be an adjunct to, but not a substitute for, personal relationships. One would have to be singularly lacking in insight not to be perturbed by awareness of playing sadist to a patient's role of masochist (as with Case 6). H. G. Jones (in Eysenck, 1960) reported the successful use of a conditioning technique in the treatment of diurnal frequency of micturition. The personal relationship between the therapist (referred to as "E") and the patient was disregarded despite the repeated assaults on her perineum required by catheterization and the repeated pelvic distensions. A very similar case was cured (Oswald, 1959b) by a conditioning technique without catheterization and success was contingent upon the existence of a special personal relationship (that present during the hypnotic trance). I would respectfully suggest that the relationship between Mr. H. G. Jones and his patient was of equal importance.

The case-report by Raymond (1956), which appears in Eysenck's book, provoked Smith (1956) to draw attention to numerous reports claiming cure of fetishism brought about by analytic techniques.

The report by Barker *et al.* (1961) prompted MacDonald (1961) to describe success in the treatment of a transvestist by psychotherapy. The cure of cat-phobia using a deconditioning technique by Freeman and Kendrick (1960) led Stevenson (1960) to draw attention to cures through hypnotherapy. Just as it would, quite evidently, be rash to discount the merits of the personal relationship of the patient to the therapist, so it would be to lose sight of the value of symptoms. Shortly before the paper by Freeman and Kendrick (1960) I began treating a case of wasp-phobia by deconditioning techniques—without success, for one cannot cuddle wasps, and the symptom enabled the patient to keep a check on her cheerful, indiscriminately amorous embezzler of a huband. The "crucial" tenet of Eysenck (1960, p. 9) that "there is no neurosis underlying the symptom, but merely the symptom itself" is unacceptable.

In this paper have been recorded both successes and failures of "behaviour therapy". Perhaps the failures arose because of some degree of sleep-deprivation or on account of the tape-loops, but the clinician who reads the book *Behaviour Therapy and the Neuroses* is first met by a stiff warning from Eysenck (1960, p. 10), that only a thorough acquaintance with modern learning theory can allow him to judge, let alone use the techniques, "It cannot be said too plainly that a superficial knowledge . . . is not sufficient." On the subject of aversion therapy, in particular, both Eysenck and Franks (Eysenck, 1960, p. 12 and p. 284), though evidently not writing from personal experience, make it plain that priority as a cause of failure must be given to the amusing ignorance of clinicians lacking "quite elementary knowledge". Forced, as I thus am, to a declaration of qualifications, only a formal education in experimental psychology and subsequent research therein at university departments other than Professor Eysenck's can be offered. Having had experience of alcohol aversion therapy, I would, however, defend those clinicians whose knowledge was brushed aside because they gave alcohol after nausea began. In the present series it was given both before and after. Offer the patient a glass of whisky when he is nauseated and what does he do? He looks at it, retches, closes his eyes in disgust, he is firmly made to drink, he retches, he drinks again, he retches and vomits, he looks with loathing at his glass, drinks again, retches, vomits, retches and retches again, and hangs in misery over his bowl of vomitus with his saliva drooling down. A picture of utter dejection. The conditioned stimulus?—the glass of whisky and the act of drinking. The unconditioned stimulus?—the miserable vomiting and searing retching on an empty stomach which follow, leading to the acquisition of a conditioned avoidance response.

SUMMARY

A method is described whereby non-schizophrenic persons can be induced to hear illusory and hallucinatory voices by causing them to listen to repetitive personal remarks. The phenomena ranged from understandable mishearing to frank hallucinosis resembling acute paranoid schizophrenia. The observations were made during "behaviour therapy", including that of two rubber-clothing fetishists and a transvestist, of which only one fetishist appears to be cured. The phenomenon of the "voices" is discussed in relation to recent reports of other workers. Some of the practical features of "behaviour therapy" are discussed and it is argued that (contrary to Eysenck's views) the personal relationship between patient and therapist cannot be disregarded.

ACKNOWLEDGMENTS

I wish to acknowledge the encouragment of the late Professor Alexander Kennedy, of Professor G. M. Carstairs and of Dr. F. J. Fish, under whom the patients were admitted. I wish particularly to thank Dr. W. D. Boyd for permitting me to describe Case 5 and Dr. P. Olley for co-operation with Case 4. Grateful thanks are due to the Matron and nursing staff of Jordanburn Hospital, especially Mr. R. Davidson. Mrs. M. Clarke and Mr. R. Baxter readily gave certain technical assistance.

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Reprinted from THE QUARTERLY JOURNAL OF EXPERIMENTAL PSYCHOLOGY, Vol. XIV, Part 3, August, 1962.

THE EEG, EYE-MOVEMENTS AND DREAMS OF THE BLIND

BY

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It was found that characteristic frontal EEG waves significantly often precede the rapid eye movements of dreaming. The rapid eye movements were absent during dream periods of three men with life-long blindness, and of two men, 30 and 40 years blind respectively, but were present during dream periods in three men blind only 3, 10 and 15 years respectively.

INTRODUCTION

In recent years, numerous American and French workers have described the invariable cyclical appearance, during continuous nocturnal sleep, of light sleep patterns in the electroencephalogram (EEG), accompanied by frequent and characteristic clusters of rapid conjugate eye movements (REMs) and dreaming (e.g. Dement and Kleitman, 1957*a* and *b*). The REMs have been claimed to represent scanning movements with respect to the dream visual imagery (Dement and Kleitman, 1957*b*). The "REM periods" with accompanying EEG picture vary in duration (20 min. is common) and four to six occur per night, together with characteristic muscle tonus changes (Berger, 1961).

Schwartz and Fischgold (1960) and Jouvet, Michel and Mounier (1960) described brief appearances of 2 to 3 c./sec. frontal EEG activity, having a characteristic "saw-toothed" wave form associated with the REMS.

We decided to investigate the hypothesis of the American workers that the REMs represent what may be called "looking-at-dream-picture" movements. First, by a closer examination of the time relations between the "saw-toothed" frontal EEG activity and the onset of individual REM clusters; secondly, by studies of the EEG and eye movements during nocturnal sleep in the blind.

Jastrow (quoted by Ramsay, 1953) studied the dream reports of persons who had become blind at varying periods after birth. Those who became blind later than early childhood maintained visual imagery but there was a progressive decline of reports of visual imagery as the individual became older. A few individuals only were said still to report some visual imagery after 40 years of blindness.

METHOD AND RESULTS

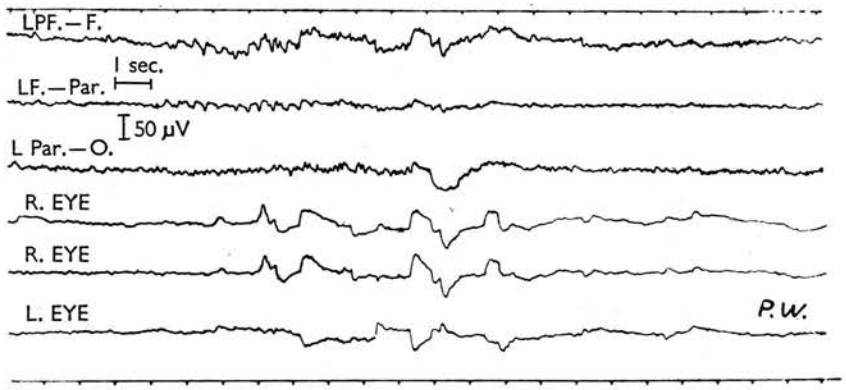
The EEG "saw-toothed" waves

In the course of other studies of all-night sleep we observed that among 36 normal persons, on whom suitably located electrodes had been used, were 23 who showed the 3 c./sec. frontal "saw-toothed" waves very prominently. It seemed clear to us that generally these brief bursts of EEG waves *preceded* individual REM clusters. In order to confirm this hypothesis, we recalled the first of our subjects from whom we had recorded the saw-toothed waves, and recorded one further entire night's sleep. The EEG was recorded using a left antero-posterior chain of silver cup electrodes, and eye movements by means of the most anterior EEG electrode and two other electrodes fixed just below the outer canthus of each eye. The diagonal and asymmetrical electrode placement had the advantage of economy, and allowed

both horizontal and vertical REMs to be recorded on the same channels. Figure 1 illustrates.

Subsequently, with the eye movement channels and then the EEG channels of the record alternately obscured, two of us (I.O. and R.J.B. respectively) independently marked the record where it was judged that each "saw-toothed" burst and each REM cluster began and ended. Figure 2 indicates that our hypothesis was confirmed;

FIGURE 1



An example of "saw-toothed" frontal activity preceding the appearance of a brief REM cluster by 3 sec.

LPF—F = left pre-frontal—frontal.
LF—Par. = left fronto—parietal.
L Par—O = left parieto—occipital.

the onset of "saw-toothed" frontal EEG waves is significantly ($\chi^2 = 229{\cdot}4$, $n = 2$, $p < 0{\cdot}001$) related to the five seconds preceding a REM cluster. This result was obtained by comparing the observed frequencies with those which would be expected to occur by chance, the latter being proportional to the total duration of each condition.

Blind subjects

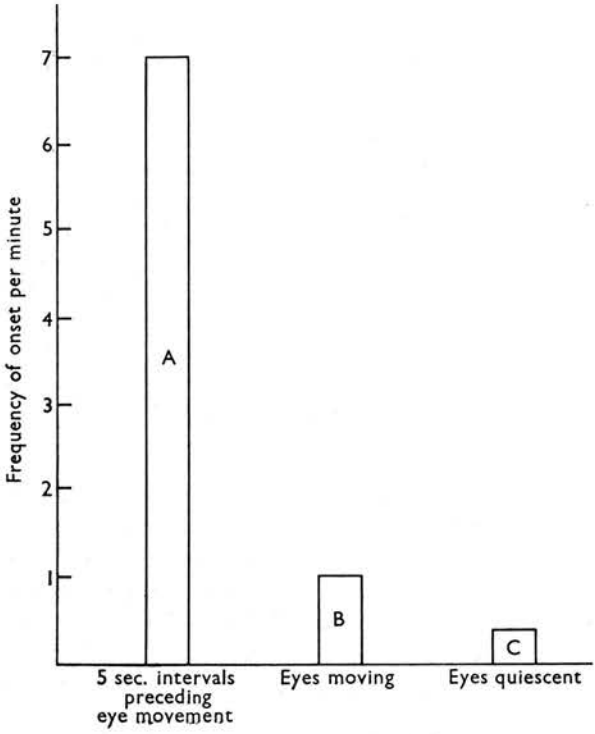
An antero-posterior chain of silver cup electrodes was used to record the EEG. A pair of electrodes was fixed at each outer canthus, to give four different electrode combinations, to enable us to recognize the occasional electrode artefact (these sometimes look like REM potentials). Eight blind male adults were studied, each having two entire nights of recording.

Three men had had life-long blindness; two, aged 25 and 30, on account of severe corneal scarring (probably resulting from neonatal gonorrhoea) and one aged 17, on account of bilateral optic atrophy since the age of 18 months or earlier. These men had no conception of visual imagery. A 60-year-old man had been blind for 30 years owing to corneal scarring, and a 52-year-old man for 40 years owing to traumatic retinal detachment and cataracts. The latter two men both stated that they had lost the ability to picture things and claimed that no visual imagery was present in their dreams. Each was first shown to produce clear electro-oculographic potentials when the eyeballs were moved voluntarily. None of these six men had REMs in their cyclically recurring light sleep periods, during which, to

judge by their subsequent reports, they nevertheless dreamed of non-visual fantasy experiences.

Three men aged 23, 33 and 40, totally blind for 10, 3 and 15 years respectively claimed still to experience visual imagery. Each had REMs in their cyclically recurring light sleep periods differing in no way from those of normal sighted people, and each when wakened during REM periods recalled visual dreams. As an illus-

FIGURE 2



Frequencies of occurrence per minute of the "saw-toothed" activity during 5-sec. intervals preceding REM clusters, during REM clusters, and when the eyes are quiescent during the cyclically recurring light sleep periods.

A: 28 instances of onset of saw-toothed activity in a total of 4 min. (namely, 5-sec. periods preceding a total of 48 REM clusters).

B: 20 instances of onset of saw-toothed activity during the course of the 48 REM clusters of total duration 20 min.

C: 21 instances of onset of saw-toothed activity during a total of 72 min. while the eyes were quiescent during REM periods.

tration, the man who had been blind for 15 years (who had particularly active REMs) described how he dreamed he was in the swimming bath, how he did the "crawl" rapidly to the other end where he could see his friend's wife ("her white skin and black costume, it was all very vivid") and splashed water teasingly over her. Of the eight blind men, only this last man had "saw-toothed" waves.

Discussion

Wolpert (1960) implies a theory of peripheral control of dreaming in the statement: "Thus REMs appear to be specific to the manifest dream content, thereby confirming the suggestion put forward by Ladd as early as 1892 that during dreaming the eyeballs move gently in their sockets, taking various positions induced by the retinal phantasms as they control the dream."

A peripheral theory of this nature does not commend itself to us and we find it difficult to reconcile our observations concerning the "saw-toothed" EEG waves with a looking-at-the-dream-pictures hypothesis. It could perhaps be argued that dreaming occurs in a series of short flashes: the frontal activity being a physiological concomitant of the commencement of each dream flash, the fantasied contents of which provoke scanning eye-movements. Dement and Wolpert (1958), however, argue strongly that dreaming is continuous. Possibly one could argue that the frontal EEG "saw-toothed" waves are indicative of a sudden change of neurophysiological status quo, which itself results in more "activity" in the dream events, so calling forth more scanning eye-movements.

A number of other theoretical objections to the looking-at-dream-pictures hypothesis have been made elsewhere (Oswald, 1962), but the presence of REMs in those blind subjects who still possessed visual imagery, and their absence in those who had either never had, or had lost visual imagery, must be considered to support the hypothesis. However, it would seem to us most economical to suppose that both the burst of EEG waves and the subsequent REM cluster are indicative of a sudden change of neurophysiological conditions, without reference to awareness of, or response to dream contents, for it is known that an artificially induced change of electro-physiological activity of the frontal cortex in the conscious human can be followed by conjugate eye-movements (Rasmussen and Penfield, 1948). It could be supposed that, owing to non-use, nervous pathways involved in the execution of conjugate eye-movements are poorly developed in those with life-long blindness, or, once established, suffer through disuse during prolonged blindness.

We should like particularly to acknowledge the encouragement and facilities given us by Professor J. Drever and Professor G. M. Carstairs.

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EFFECTS OF SLEEP DEPRIVATION ON BEHAVIOUR, SUBSEQUENT SLEEP, AND DREAMING

BY

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Reprinted from

THE JOURNAL OF MENTAL SCIENCE

Vol. 108, No. 455, July, 1962

EFFECTS OF SLEEP DEPRIVATION ON BEHAVIOUR, SUBSEQUENT SLEEP, AND DREAMING

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Introduction

In the course of the past few years, a series of related studies (Aserinsky and Kleitman, 1955; Goodenough *et al.*, 1959; Wolpert and Trosman, 1958) has demonstrated beyond doubt the association of normal dreaming with the appearance of rapid, binocularly synchronous eye-movements. It has also been claimed that the rapid eye-movements (REMs) represent scanning movements made by the dreamer as he "watches" the visual events of the dream (Dement and Kleitman, 1957a; Dement and Wolpert, 1958). The REMs are absent during dreaming among those with life-long blindness, but are retained for some years by those whose blindness arises later than childhood (Berger *et al.*, 1962a). In a study of undisturbed nocturnal sleep by Dement and Kleitman (1957b) periods of eye-movements were observed to occur fairly regularly at about 90-minute intervals throughout the night in association with the lightest phases of cyclic variation in depth of sleep, as indicated by the electroencephalogram (EEG). These REM periods had a mean duration of about 20 minutes, and 4–6 occurred per night.

The hypothesis has recently been put forward by Dement (1960a) that a certain amount of dreaming each night is a necessity. Following a series of nights of "dream deprivation" in which subjects were prevented from dreaming by being deliberately awakened at the commencement of their REM periods, Dement observed a marked increase in total dream time and percentage dream time on subsequent recovery nights of undisturbed sleep, as compared with that on a number of base-line nights prior to dream deprivation. He also reported that "dream deprived" subjects became unhappy and ill at ease. We understand that certain independent research groups in the U.S.A., in work as yet unpublished, have confirmed Dement's findings.

The aim of the present study was to compare the percentage dream time during nocturnal sleep before and after four consecutive nights without sleep It was predicted by the authors when beginning the experiment that the percentage dream time would be markedly reduced after sleep deprivation and would then gradually return to the base-line levels on consecutive recovery nights, and that the sleep loss would manifest itself as an increase in percentage time spent in deep sleep on the initial recovery night. However, according to Dement's hypothesis, it would be predicted that the acquired dream-deficit following sleep deprivation should be compensated for by an increase in dreaming on subsequent recovery nights compared with that on the base-line nights.

The visual, auditory and somatic hallucinations, the intrusive thoughts, the bizarre verbal constructions with queer word play and neologisms, the paranoid delusions and failure of discrimination between fantasy and reality that occur in normal bedtime drowsiness and in sleep-deprived persons have been reviewed

(Oswald, 1962). Tyler (1955), Bliss *et al.* (1959), Brauchi and West (1959) and Morris *et al.* (1960) in particular have discussed the similarity of the thinking and perception of their sleep-deprived subjects to those of schizophrenics. In the present experiments, the opportunity was therefore taken to record any psychotic features of the subjects' experiences. These proved surprisingly frequent, and only representative examples are given below.

Method

The subjects, who were paid, were six healthy male medical students aged 21–24. They were informed of the nature of the experiment on personal interview.

Briefly, the experimental procedure was as follows. The experiment consisted of four stages:

Stage 1. 2–3 adaptation nights of sleep.
2. 4 base-line nights of sleep.
3. 4 nights of sleep deprivation.
4. 4 recovery nights of sleep.

The entire experiment was performed using subjects in pairs.

1. *Adaptation Nights*

The subjects came to the laboratory at their normal bedtime and on the initial nights accustomed themselves to sleeping in the novel situation.

On the final adaptation night, the conditions represented those of the subsequent recording nights. Electrodes were attached to the head and the leads plugged into the headset placed between the parallel beds.

2. *Base-line Nights*

Each pair of subjects reported to the laboratory one hour before their usual bedtime. The EEG was recorded by means of an anteroposterior chain of silver-cup electrodes affixed with collodion, and eye-movements by means of the anterior EEG electrode and two other electrodes fixed just below the outer canthus of each eye with sticking plaster. The diagonal and asymmetrical electrode placement had the advantage of allowing both horizontal and vertical REMs to be recorded on the same channels. Two eye-movement and two EEG channels were recorded from each subject on an 8-channel electroencephalograph situated in a room adjacent to the bedroom.

3. *Sleep Deprivation Nights*

The subjects were instructed to remain awake at all times and were under close supervision by one of us (R.J.B.) throughout the night, and by the other (I.O.) during the day. They were stimulated to keep awake at night by playing cards, "Monopoly", table tennis, billiards, etc. During the day, they were constantly employed on simple tasks. The repeated introduction of variety into the activity of such subjects is essential for the maintenance of wakefulness.

4. *Recovery Nights*

These exactly duplicated the base-line nights except for the first recovery night, when the subjects retired to bed in the early hours of the evening and were

allowed 12 hours sleep in comparison with 7–8 hours on the base-line and subsequent recovery nights. They were awakened at approximately the same time the following morning, so that they spent the same number of hours awake during the day before the second recovery night as on all other recording nights.

Instructions

The subjects were requested not to sleep at any time throughout the experiment, other than on the designated recording nights. They were also asked not to consume alcohol within the four hours before retiring and any consumed previous to that to be moderate in quantity.

Results

The results may be considered in relation to nightly dream time, depth of sleep, and abnormal behaviour during sleep deprivation.

1. *Nightly Dream Time*

The times of onset and termination of dreaming were judged from the records by the first and last rapid eye-movements of a REM period. The relevant data are summarized in Table I.

Base-line Nights. Mean sleep time for the 24 nights was 7 hours 26 minutes, and the mean percentage dream time (mean total dream time to total sleep time $\times 100$) was 22·5 (Standard Deviation=1·48).

Recovery Nights. Since the subjects were allowed 12 hours sleep on their first recovery night, the data given in the fourth column of Table I refer to the first part of the night corresponding to the mean sleep time of the base-line nights for each individual. As can be seen from the Table, the mean percentage dream time was 7·4, which is roughly 30 per cent. of the group

Table I

Subject	Mean and Range Base-line Nights		Recovery Nights % Dream Time and Total Sleep Time					
	Total Sleep time	% Dream time	I	II	III	IV	I (Whole Night)	II Equalized
S.A.	7h. 38m. 7h. 17m.–7h. 52m.	22·5 20·6–25·6	6·3	24·6 7h. 47m.	20·0 7h. 44m.	16·4 7h. 49m.	14·4 12h. 0m.	24·6
A.L.	7h. 7m. 6h. 49m.–7h. 15m.	22·7 18·8–25·4	—	23·1 7h. 47m.	27·1 7h. 41m.	28·1 7h. 49m.	11·9 12h. 0m.	23·0
A.M.	7h. 26m. 7h. 12m.–7h. 40m.	23·4 22·0–24·7	10·1	30·0 7h. 40m.	31·7 7h. 50m.	28·0 7h. 50m.	23·8 11h. 50m.	28·0
J.D.	7h. 42m. 7h 17m.–8h. 00m.	20·4 17·5–22·7	5·4	24·0 7h. 39m.	26·8 7h. 57m.	22·5 7h. 51m.	12·4 11h. 55m.	24·0
K.M.	7h. 30m. 7h. 21m.–7h. 33m.	21·2 18·7–24·5	8·0	28·4 7h. 49m.	21·6 7h. 44m.	19·3 (16·4 B.L.) 5h. 22m.	22·9 11h. 48m.	28·4
C.S.	7h. 14m. 7h. 7m.–7h. 21m.	25·0 22·2–29·9	14·7	35·0 7h. 51m.	34·3 7h. 43m.	14·7 (19·6 B.L.) 5h. 20m.	24·0 11h. 55m.	31·3
Means	7h. 26m.	22·5	7·4	27·5	26·9	23·8 (last 4 subjects)	18·2	26·9
Level of Significance ..			·1%	5%	N.S.	N.S.	N.S.	2%

base-line mean. This marked decrease was highly significant ($P < \cdot 001$) on the t-test for significance of mean difference (which was used in all statistical calculations).

Taking the whole of the first recovery night, the mean percentage dream time was 18·2, which was still less than that for the base-line nights, although the amount of dreaming increased considerably towards the latter part of the night.

On the second recovery night, the mean percentage dream time increased to 27·5, which represents a 20 per cent. increase of dream time over the base-line mean. This result was significant ($P < \cdot 05$).

Although the mean percentage dream time on the third and fourth recovery nights was still greater than on the base-line nights, the difference is not statistically significant.

The proportion of sleep spent in dreaming in normal persons increases with the duration of sleep. In order to test whether the significant increase in dream time on the second recovery night might not be due to an overall increase in total sleep time (since all subjects except J.D. slept longer on their second recovery night than the mean times for their base-line nights), the percentage dream time was recalculated (last column of Table I) by ignoring the final part of the record in all subjects which was in excess of their mean sleep time on the base-line nights. Although the group mean percentage dream time was reduced to 26·9, the increase over that of the base-line nights had a higher level of significance ($P < \cdot 02$) since the variance was reduced by this correction.

The figures given for the fourth recovery night of subjects K.M. and C.S. require comment. Unfortunately, the recording apparatus broke down in the latter half of the night and the percentage dream time figures are consequently reduced. The figures given in brackets are the mean percentage dream time for the same duration of sleep on their base-line nights, and these figures were used in the statistical caculations.

On consideration of the sleep pattern, the increase in percentage dream time on the second recovery night appeared to result from an increase in the frequency of the sleep cycle, rather than from increased dream duration or the earlier appearance of rapid eye-movements and dreaming owing to more pronounced lightening of sleep in the initial hours of the night.

2. *Depth of Sleep*

The depth of sleep as characterized by the EEG on the first recovery night was compared with that on the fourth base-line night, for each subject, adopting the criteria of five stages, "A" to "E", of increasing depth of sleep as defined by Loomis *et al.* (1937). Since the first recovery night was of greater duration than the fourth base-line night in all subjects, the final part of the night's sleep in excess of that on the base-line nights was not included in the analysis, so that total sleep times were equated.

The mean times spent in stages B, C, D and E were transformed to mean percentages of total sleep time, and the results are represented by Figure 1. It is clear that the decrease in time spent in dreaming in the B-stage, and also the decrease of C-stage sleep on the first recovery night, is made up by a corresponding increase in time spent in E-stage sleep, as compared with the fourth base-line night. On statistical analysis, the decrease in B-stage and increase in E-stage sleep were highly significant ($P < \cdot 001$) and the decrease in C-stage sleep significant ($P < \cdot 05$).

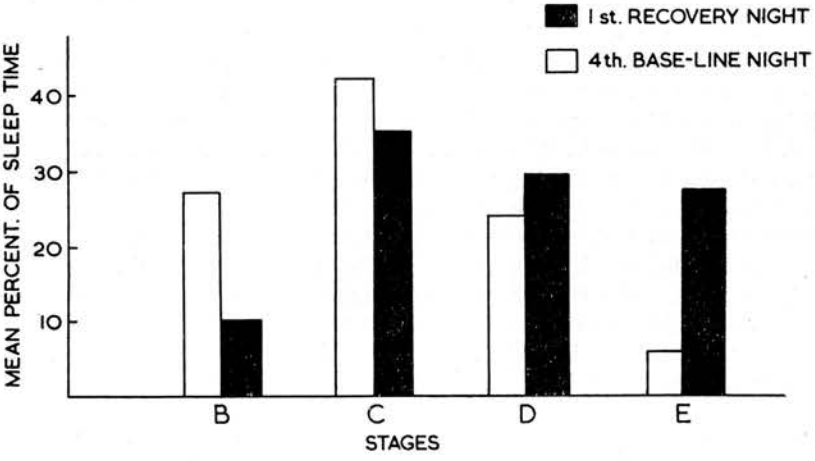

FIG. 1.—Mean percentage of total sleep time spent in Stages B, C, D and E on fourth base-line and first recovery nights. Note the increase in time spent in deep sleep (EEG stage E of sleep) following sleep deprivation.

3. "*Psychotic*" *Changes During Sleep Deprivation*

All subjects experienced changes of visual perception of surfaces. Wallpaper patterns seemed to move and flow, swirling vapour or cobwebs or shimmering bubbles seemed often to cover the floor, their hands, or the table, and one man once spent half a minute carefully kicking at the cobwebs which appeared to cover a carpet. Two saw crumbs on the table-cloth running about like insects.

Three had hallucinations of women peering at them. One (S.A.) had these experiences frequently after 60 hours, many being in broad daylight. The women were almost all unpleasant old women, who appeared to be talking about him. They would vanish (sometimes the body before the face) as he drew near, but after passing he saw them again when he turned round.

Sometimes the subjects addressed those present as if believing they were speaking to quite different persons, sometimes actually using the wrong name. On many occasions the subjects would speak or write what appeared to be dream contents. When given simple tasks such as addressing letters from a list, they made numerous mistakes (e.g., writing "West Looking" for "West Lothian"); or when asked to turn over all-night EEG recordings and write down the time every two minutes (i.e., every three double pages) they often wrote down absurd phrases instead of the time (e.g., C.S. wrote, "Batting by one", "cormial brier", "adorable"). While C.S. was working through an EEG record, he suddenly made the irrelevant remark, "Who to begin". Soon afterwards, he kissed the EEG paper. When asked about this, he said he must have been dreaming about his girl friend (who was currently denying him her hand in marriage). He was asked to write a description, but, as so often was the case, absurd elements quickly intruded. He wrote, "Leant forwards and downwards to plant a kiss upon the unmaried letters. £Coo hch". (Note the evident associations, EEG paper—letters—hch).

Each pair of subjects was sometimes sent on brief shopping errands by day. Once on return A.M. spoke at length to one of us about a "severe accident", and persisted on this theme despite replies. Speech tended to be rather mumbling on these occasions. When playing a card game in the night, A.M. remarked suddenly, "Too many schoolboys and schoolgirls working outside their school

lessons. It is a result of the decadence of this country . . . kill too many birds with one stone. Got to keep warm . . . grab my pubic hairs . . . go from one to the other." When questioned about all this, he muttered about "certain fleeces" and jumping from one hair to another. When asked on another occasion about a paint brush he was holding, he replied that the National Trust for Scotland were organizing an island visit. When J. D. said, "I can't find a towel", A.M. replied "Have you got diarrhoea?" Replies to questions would sometimes start normally, but finish oddly. A.M., describing this, said he felt his "own normal personality in conflict with another which seized upon some unrelated, irrelevant thought and gave voice to this thought in the middle of a conversation".

Another example typical of dream-thinking occurred when R.J.B. was chatting with A.M. about some experimental work. Where normally the phrase "first in the field" might be used, A.M. said, "That is all right so long as you are first on the green." He had a visual image of three golf balls on a green, one played by R.J.B.

Three subjects had paranoid delusions. K.M. one night behaved and spoke oddly for an hour and afterwards admitted that he had thought one of us (R.J.B.) was some sort of captor or interrogator. At the time, he spoke of R.J.B. as an "exquisitor", which he later explained meant to him an inquisitor able to inflict exquisite pain. A.M., when starting to play a card game, volunteered the statement that he intended to hire a bodyguard to protect him from "them". On being questioned about "them", he said, "They are the ones who caused all the trouble in the last year."

The most elaborate and firmly held delusions were those of A.L. After 96 hours of sleep deprivation, he experienced hallucinatory voices while a water tap was running. He turned off the water "to hear them better", but then they ceased. Later that morning he and S.A. went shopping with one of us (I.O.). Walking along a main street, A.L. was walking behind S.A., peering and pointing at the latter's jacket. He said he saw handwriting on it. A few minutes later he insisted on his companion taking off his jacket in the street (which he did) for a closer inspection. It was explained to him that such experiences occurred during sleep-deprivation. However, from that point on he elaborated a delusional system which he did not divulge till seven hours later and not fully till the next day, when he wrote nine pages of description.

It was planned that he and his companion should appear on the television that evening. He believed he must have been given a hallucinatory drug in order that he should have something interesting to recount on the television. He recalled that he had been told to drink up his mid-morning coffee before the shopping expedition and decided the drug must have been in the coffee. At lunch "we were put at a table well away from the rest . . . I said, 'What do they think we are, bloody pariahs?' I then thought I heard someone two or three tables away talking about the word pariah and its derivation. I decided that I must have been treated so that although I thought I spoke in a normal voice, I was, in fact, shouting. During the afternoon I was doubtful what I should do about this information I had, as I didn't want to tell . . ." When he saw I.O. speak to another doctor at a lunch-table, this was an indication that his realization of the drugging had been discovered. The waiter brought two jugs of water to his table, which indicated that further drugs had been administered in the table-salt and "I experienced a rather bitter taste in my mouth".

In the afternoon the subjects were left temporarily in the charge of a female secretary who was armed with several small shopping lists. After each errand, it was emphasized that they should return to her room. A.L. was very puzzled by

this, but concluded it was because the hallucinatory handwriting on the jacket was much clearer in the fluorescent lighting in that room. Once in this time he unsuccessfully sought I.O. out and once made an abortive attempt to telephone —in order to reveal that he knew about the drugs.

In the late afternoon I.O. handed some papers over to R.J.B. and wrote a pencil note on one. To A.L. this was a note stating that he had discovered about the drug. Later in the evening, however (see below) he concluded it must have been a message stating that his companion S.A. was mentally unbalanced. He was puzzled that I.O. did not acknowledge repeated hints that he (A.L.) gave to indicate his discovery.

They were driven to Glasgow in a television company car. A.L. felt very frightened, for R.J.B. was questioning S.A. about his dreams, and the realization dawned that R.J.B. was, in fact, a psychiatrist (and not a psychologist after all) who was taking S.A. away to lock him up after first hypnotizing him, for S.A. kept moving his limbs at the command of R.J.B. (actually this was to keep S.A. awake). A word-game was played to help maintain wakefulness, but A.L. (as he subsequently wrote) "was very wary of this because . . . of producing a word of special significance, like a Freudian slip". When given the word "train", he replied "Glasgow", for it instantly dawned on him that S.A. must be about to be locked up for having been responsible for the fires on the Glasgow electric trains withdrawn from service some months before.

He now became more frightened, for it dawned upon him that he and not S.A. was to be locked up. R.J.B. noted the strange, fixed stare on his face and received a large number of evasive and queer answers to questions ("discovering the unknown"; "the guilty one"; "the characters are different".). They reached the television studios, which A.L. took to be the hospital. He then confessed his fears and beliefs, was taken indoors and, when shown the cameras etc. his delusions seemed to evaporate. He was quite normal the next day after the first recovery night.

Discussion

There are grounds for identifying the cyclical changes in depth of human sleep with similar but faster cyclical changes observed in the cat. In both species, rapid eye-movements occur when the EEG signs of light sleep appear together with irregular respiration and heart rate, and relaxation of skeletal musculature (Jouvet *et al.*, 1960; Berger, 1961). There is reason to believe that dreaming also occurs in cats at these times (Dement, 1958). The term "paradoxical phase" has been used to describe this phase of sleep, for it has special neurophysiological characteristics and has been called "hind-brain sleep" by Jouvet *et al.* (1960).

Following the original report by Dement (1960a), discussion (Ullman, 1960; Dement, 1960b) has centred upon the interpretation of Dement's findings from consideration of psychic or physiological mechanisms of stability. The observations of Dement (1960a) were explained by him in terms of dream deprivation. An explanation in terms of deprivation of the "paradoxical phase" of sleep would be one alternative. Another alternative was proposed by one of us (Oswald, 1962), but that alternative could not account for the results of the present work, which were contrary to our prediction, and which in fact were consistent with what would have been predicted if Dement's views had been accepted. It could be argued, of course, that sleep deprivation interferes with our acquired 24-hour rhythms so that sleep deprivation might be followed by a tendency to more light sleep and dreaming. In other investigations (Berger *et al.*

1962b), we have found clinical doses of barbiturates to reduce dreaming time. If this is true when barbiturates are taken over very prolonged periods, one would expect definitely adverse effects if dream deprivation were harmful, as Dement (1960a) suggests it to be. We are not, however, aware of serious harmful effects.

There seems little justification for arbitrarily isolating one psychic or physiological component of the total process and propounding a "need" for it. One might as validly propose that there is a "need for rapid eye-movements".

Our observations of increased depth of sleep following sleep deprivation are compatible with those of Marbach and Schaff (1960), who found a decrease of body movements during sleep following one night of sleep deprivation. However, observations during the paradoxical phase in cats, of elevated auditory thresholds (Dement, 1958); of raised threshold to reticular formation stimulation (Benoit and Bloch, 1960; Jouvet *et al.*, 1960), and behavioural arousal with EEG slow waves (Horovitz and Chow, 1961) to reticular formation stimulation, suggest the need for definitive criteria when using the terms "deep sleep" and "light sleep". Whatever the need for sleep in which dreaming occurs, our results demonstrate that deep sleep, in the more traditional EEG sense, takes priority after sleep deprivation.

A knowledge of the literature had not prepared us for such frequent psychotic features. These may well have been so much more apparent to us because the pairs of subjects literally lived continuously with one or other of us, whereas most other writers appear to have relied heavily on relays of nurses and other assistants to keep large groups of subjects awake, and the conclusions of Morris *et al.* (1960), for instance, were primarily based on a pair of clinical interviews with each subject. A sleep-deprived person can usually "pull himself together" sufficiently well to appear practically normal provided he has to do so only for a short period.

Sleep-deprived persons can maintain their cerebral vigilance at normal waking levels briefly, but there is an insistent downward drift of cerebral vigilance, accompanied by changes in thinking and perception comparable to those of dreams. Kraepelin (1906) wrote the most extensive paper on the peculiarities of dream-speech constructions and was intrigued by similarities to dementia praecox. It is remarkable that, while a great deal of interest has centred on drugs which produce experiences reminiscent of those described by schizophrenics, similar states produced without drugs by sleep deprivation alone have been comparatively neglected. The cases described here illustrate the verbal constructions of lowered cerebral vigilance, and how, as in the case of A.L., it is not necessary to give any drugs whatever to produce hallucinations and elaborate paranoid delusions.

Summary

The EEG and eye-movements during nocturnal sleep were recorded from six males on four base-line nights and four recovery nights following 108 hours of sleep deprivation.

On the first recovery night there was a significant increase in the mean percentage of total sleep time during which EEG signs of deep sleep were present, associated with a significant decrease in the mean percentage of total sleep time spent in dreaming (determined by duration of rapid eye movement periods) on comparison with the base-line nights.

On the second recovery night there was a significant increase in mean dream time percentage compared with that on the base-line nights.

The results are discussed in relation to theories of a "need" for dreaming.

Hallucinations, paranoid delusions and other abnormal behaviour during sleep deprivation are described.

ACKNOWLEDGMENTS

We wish to thank Professor G. M. Carstairs and Professor J. Drever for their encouragement and facilities extended to us. The work was aided by a grant from the Alexander Kennedy Research Fund.

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From Proceedings of the Royal Society of Medicine,
1962, Vol. 55, pp. 910 - 912.

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Sleep Mechanisms: Recent Advances

Recent advances in knowledge about sleep have depended heavily on the electroencephalogram, and in the last ten years much effort has centred on electrophysiological studies of the function of the brain-stem reticular formation. It is now known that, besides impulses branching off from the main sensory paths, many other factors govern the level of excitement of the reticular formation (Oswald 1962). Bonvallet *et al.* (1954) in Paris demonstrated the first known afferent inflow to damp down reticular formation excitement and promote sleep, namely the afferent inflow from carotid sinus stretch receptors. This illuminated the rare clinical cases of attacks of impaired consciousness from stimulation of abnormally sensitive carotid sinuses (Ferris *et al.* 1935, Engel 1959).

It seems also that the likelihood of snatching a daytime nap depends on the carotid sinus, which would be expected to distend slightly when atmospheric pressure fell. Raboutet *et al.* (1959) analysed the conditions favouring the appearance of brief spontaneous sleep during 642 routine electroencephalographic (EEG) examinations of healthy males. There was little or no relation to temperature or humidity, but a significant relation to atmospheric pressure. The lower it fell, the more likely was the individual to take a nap.

It is also clear that the cortex is an important cause of reticular formation excitement. It was found that meaningful words were significantly more potent in arousing sleeping humans than identical physical stimuli made meaningless by altering the order of their components in a tape recording. Such complex auditory discriminations between speech sounds depend on cortical action, subsequent to which impulses must have passed from the cortex to excite the reticular formation and promote arousal (Oswald *et al.* 1960).

The reticular formation exerts a subtle and constantly varying control over cerebral vigilance, according to the demands of the moment. Light sleep and wakefulness can alternate several times in the space of 10 seconds (Oswald 1959, 1960).

Paradoxical Sleep

French workers (Jouvet *et al.* 1960, Jouvet 1962) recorded from implanted electrodes in the brains of sleeping cats. The high voltage slow waves and spindles of sleep from the cortex were also recorded from the mid-brain, but wakefulness rhythms often persisted in the pontine reticular formation. Repeatedly during sleep there would be a sort of abrupt reversal lasting several minutes – slow waves and spindles from the pons, rhythms like those of wakefulness from the cortex, coupled with a sudden disappearance of tone from the nuchal muscles. Although these trunk muscles are more relaxed during this 'paradoxical' sleep, as Jouvet has called it, cats during it twitch their whiskers and tails and appear to be dreaming, their eyeballs making frequent rapid movements (Dement 1958).

In man, although at first I, for one, interpreted paradoxical sleep as light sleep (Oswald 1962), it is now clear that we must acknowledge the existence of *two kinds of sleep*. Recurrently through the night, about every one and a half hours, for 10–50 minutes at a time the orthodox sleep gives way to paradoxical sleep with rapid eye movements. Dement & Kleitman (1957*a*, *b*) and numerous others since have established that when people are awakened from paradoxical sleep with rapid eye movements they usually say they have just been dreaming, whereas at other times of the night they will rarely report dreams.

The EEG picture of human paradoxical sleep resembles that of drowsiness before falling asleep. However, in drowsiness the eyeballs make gentle rolling movements, whereas in paradoxical sleep the movements are rapid and jerking in character. Furthermore, in most of the adult population

'saw-toothed' waves of characteristic appearance are seen during the paradoxical phase. Each burst of these waves generally precedes a burst of rapid eye movements (Berger *et al.* 1962); they are generally frontal in distribution, but in some people extend all over the head and show a phase reversal at the vertex. The heart rate and respiration become irregular.

We were trying to record evidence of dream-talking by recording with surface electrodes over the extrinsic laryngeal muscles when my colleague, Berger (1961), noticed the sharp decrease of muscle tone whenever paradoxical sleep began. This fall in muscle tone over the front of the neck in man we have found invariable, just as over the back of the neck in cats. In company with a momentary burst of 'saw-toothed' waves it can precede the other signs of onset of paradoxical sleep by half a minute. All other human muscles appear to relax so greatly during sleep that with surface electrodes we cannot detect any further decline with the paradoxical phase, even in post-encephalitic parkinsonism.

It seems that as in cats, so also in humans, during paradoxical sleep in which the individual is conscious (if only of his dream-world) some powerful forces cause muscular relaxation. We are reminded of those nightmares in which the dreamer feels the need to escape from some terrifying event, struggles to move, yet finds himself paralysed, his body seemingly divorced from his conscious efforts. The possible relation to the occasional sleep-paralysis of both the normal and the narcoleptic is clear. Jouvet (1962) from studies of cats has written of 'hind-brain sleep' (with something akin to 'fore-brain wakefulness'). We may wonder whether the disorder of the narcoleptic's sleep mechanisms may include not only orthodox but paradoxical sleep – his cataplectic attacks being episodes of 'hind-brain' sleep.

Dement & Wolpert (1958) reported findings they interpreted as indicating that the rapid eye movements were related to dream content, that the dreamer was, as it were, looking at the 'dream pictures'. We studied the all-night sleep of blind men (Berger *et al.* 1962). Men blind for only a few years still had visual experiences in their dreams and still had the rapid eye movements. Two men blind for thirty and forty-two years respectively who said they no longer were able to 'picture' things, had no rapid eye movements during their dreams. Likewise, three men blind all their lives had no rapid eye movements during their dreams. Such blind men dream during their paradoxical sleep and describe various fantasy adventures, but do not 'see' things in their dreams. These findings support Dement & Wolpert's theory.

Some people awakened from paradoxical sleep will say they have been asleep and dreaming; others, that they were awake and 'thinking' (Goodenough *et al.* 1959). An individual may not call it a 'dream' unless there were bizarre features about the fantasy.

Depressive Insomnia

Psychiatrists commonly have patients who say they have been recurrently awake and 'thinking' in the night – particularly those suffering from 'endogenous depression'. Are such people really awake or do they spend a lot of time in paradoxical sleep? If awake, is it at the expense of orthodox or of paradoxical sleep? Do they perhaps wake up instead of dreaming? After all the EEG signs of paradoxical sleep are of light orthodox sleep.

We studied patients selected as suffering from typical depressive illnesses (301·1 in the International Classification), who had not been receiving hypnotic drugs, and compared their sleep on four successive nights with that of normal controls matched for sex and age, both with and without heptabarbitone 400 mg as a hypnotic (Oswald *et al.* 1963). The distribution of nocturnal awakenings among the depressives revealed nothing to suggest that they awakened instead of dreaming. Awakening seemed to occur randomly and, even when they slept very little, some sleep was the paradoxical or dreaming type.

Diaz-Guerrero *et al.* (1946) claimed that depressed patients spend an excess of the night with an EEG low voltage sleep pattern – which raised the question whether they might spend an abnormal time in paradoxical sleep. We found no evidence of this, but found that paradoxical sleep was significantly shortened by the heptabarbitone. The same workers had claimed that depressives shifted the depth of their sleep abnormally frequently. We did not find this difference, but found that the drugs caused a significant fall in the frequency of shifts of sleep depth. The frequency of movements during the night as a whole was significantly reduced by the drugs.

When the early morning hours in which, by tradition, the depressed patient lies awake were considered, then both as regards the differences between patients and controls and the effect of the drugs the EEG discriminated at highly significant levels, though body movements did not. The patients were awake far more than the controls, and heptabarbitone decreased time awake.

The Need for Sleep

There is evidence that we need paradoxical sleep. Dement (1960) woke up volunteers whenever during the night their periods of paradoxical sleep began. On succeeding nights they tended more and more to pass into that kind of sleep, had to be wakened more and more often, and when finally allowed undisturbed nights spent an

abnormal proportion of time in paradoxical sleep. Other American workers have confirmed this. At first our scepticism was aroused by this 'dream-deprivation' work, so my colleague, Berger, proposed the study of the effects of total sleep deprivation with, *ipso facto*, 'dream-deprivation'. Recordings were first made while six subjects slept normally on four nights each. Then we kept each awake one hundred and eight hours. Then their next four nights' sleep was recorded. On the first of these nights, as expected, paradoxical sleep was greatly reduced, but on the second night, contrary to our prediction, the proportion of the night spent in paradoxical sleep was significantly greater than on the earlier baseline nights, and was again increased on the third and fourth nights. It was as if what they had lost they made up for later (Berger & Oswald 1962). On their first recovery nights these subjects had spent a great part of the time in orthodox deep sleep with high voltage EEG slow waves (the E stage), and it is clear that when we have gone short of sleep it is this old-fashioned kind of deep sleep that takes priority. Such highly sleep-deprived people, when allowed to sleep, take only about twelve to thirteen hours sleep and then feel nearly normal, as if the restitutive properties of the E stage of sleep with its high voltage EEG slow wave picture is greatest.

Three years ago two people in our department lived a forty-eight-hour life, going to bed only every other night and then sleeping as long as they liked. After a month, one was fairly well adapted to it. Both normally slept eight hours each night; on their new routine they both, throughout, took not sixteen hours in each forty-eight, but only eleven and a half hours sleep in each forty-eight. This raises the unanswerable problem, what is the restitutive function of sleep?

Acknowledgments: I should like especially to express my thanks to my colleague, Ralph Berger.

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Reprinted from Science, August 24, 1962, Vol. 137, No. 3530, page 601

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Eye Movements during Active and Passive Dreams

Abstract. Independent confirmation is offered that the amount of rapid eye movement during dreaming is associated with the dream content.

It is now recognized that there exist two different and alternating categories of sleep. One has been called "hindbrain sleep" by Jouvet (*1*) and is accompanied by a low-voltage and fairly fast electroencephalographic (EEG) pattern, relaxation of certain neck muscles in cat (*1*) and human (*2*) and, perhaps most strikingly, by bursts of conjugate, rapid eye movements with subsequent recall by the subject that he has dreamed (*3*). Dement and Wolpert (*4*) reported evidence supporting their hypothesis that these eye movements were directional responses to the events of the dream. Yet rapid eye movements persist in cats after decortication (*1*). Also the fact that characteristic saw-toothed electroencephalographic waves tend to precede each burst of rapid eye movements appeared to us to cast doubt on the hypothesis; the presence or absence of these eye movements during "hindbrain sleep" of blind men, according to the retention or absence, respectively, of visual imagery (*5*), would support the hypothesis. One of us (*6*) has criticized Dement and Wolpert's report but is now pleased to report confirmation of one of their findings, namely a relation between profuse eye activity and an active dream fantasy.

One of us (R.J.B.), for an entirely distinct purpose, awakened eight volunteers from periods of rapid eye movement on 103 occasions during 37 nights. Dream recall occurred in 89 instances and was recorded on magnetic tape. The dream reports were subsequently all presented to the other of us (I.O.), who had never been present during the nocturnal recording sessions and who had never seen the relevant electroencephalographic or eye movement records. He classified the dream reports as "active" or "passive" according to the nature of the events described, and especially if he felt such events would have been accompanied by many shifts of gaze, had they occurred in real life.

Subsequently R.J.B. assigned code numbers to each electroencephalographic and eye movement record and presented each to I.O., who was entirely ignorant of the dream to which each record was related and distinctly skeptical of the likelihood of the association eventually found. The eye movement periods were classified by I.O. as "active" or "passive" according to the frequency and size of the eye movements which occurred throughout each 10 to 20 minute period prior to the time the subjects had been awakened, although the later in the period the movements did or did not occur, the greater the weight he attached. The whole set of records was then inspected again in a different order by I.O. and classified a second time. R.J.B. then selected the records of the 22 instances where divergent judgments had been made, and I.O. made a final classification of these periods of rapid eye movement.

The code was then broken. Fifty dream reports had been classified as "active" and in 42 instances the relevant period of rapid eye movement had been judged "active." Thirty-nine reports had been classified as "passive" and in 23 instances the relevant period was judged "passive."

It is therefore confirmed that there is a significant association ($\chi^2 = 16.18$: $P < .001$) between the nature of the dream content and the amount of movement of the eyes.

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* Medical Research Council Grantee.

13 July 1962

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MELANCHOLIA AND BARBITURATES: A CONTROLLED EEG, BODY AND EYE MOVEMENT STUDY OF SLEEP

BY

IAN OSWALD, RALPH J. BERGER, R. A. JARAMILLO, K. M. G. KEDDIE, P. C. OLLEY and G. B. PLUNKETT

Reprinted from

THE BRITISH JOURNAL OF PSYCHIATRY

Vol. 109, No. 458, January 1963

Brit. J. Psychiat. (1963), **109**, 66-78.

Melancholia and Barbiturates: A Controlled EEG, Body and Eye Movement Study of Sleep.

By IAN OSWALD, RALPH J. BERGER, R. A. JARAMILLO, K. M. G. KEDDIE, P. C. OLLEY and G. B. PLUNKETT

Insomnia is widely accepted as a leading feature of that illness designated by the number 301·1 in the International Classification of Diseases. The name given varies—"endogenous depression", "manic-depressive psychosis/depressive type", "melancholia" or "depressive illness". The present generation of laymen (not to say some psychiatrists) use the word "depression" so freely in order to describe an unpleasant, unhappy mood, whether lasting or transient, that one may prefer the term *melancholia*, or at least "depressive illness", to signify that condition which justifies the number 301·1. The adjective "endogenous" implies for many that environmental stress plays little or no part in the onset of the illness; we cannot accept that this is always so. The term "reactive" is used in several ways, generally to indicate that the state of unhappiness arises out of some external circumstance and that it would end when circumstances improved or the individual accepted the realities of practical life. The patients used in the present study were suffering from an illness of a kind which we believe may sometimes develop in the absence of severe environmental stress, while in others it may be clearly provoked by circumstances, but the illness, as it develops, may take on a form which becomes largely independent of the environmental circumstances and may continue even when the provoking factors are past; it has become an *autonomous melancholia*. It will be apparent that by autonomous melancholia we mean an illness shown by clinical experience to respond especially well to electroplexy.

In the electroencephalogram or EEG, we today possess the best available tool for the study of insomnia and all-night sleep. The demands on labour and paper of such studies are heavy. The fact that in this study only six patients have been used and that nevertheless a page by page analysis of no less than 13 miles of paper was required, serves to explain why no adequate EEG study of this kind appears hitherto to have been undertaken.

In the last few years, there have been fundamental advances in our knowledge of nocturnal sleep, arising out of the initial work of Dement and Kleitman (1957a) and their colleagues. These advances, which have been reviewed (Oswald, 1962a), concern the presence of cyclical changes in the character of nocturnal sleep. About every one-and-a-half hours, the EEG changes to a low voltage pattern containing some alpha rhythm, but also, in many people, saw-toothed waves (Fig. 1) of unique appearance (Schwartz and Fischgold, 1960; These waves usually (Berger *et al.*, 1962) immediately precede a burst of rapid conjugate eye movements. These events continue for some 10–50 minutes before the EEG changes again to the traditional sleep pattern of spindles and high voltage slow waves with ocular repose.

The periods of sleep with low voltage EEG and rapid eye movements (REMs) have been termed "REM periods" or the "paradoxical phase" (Jouvet *et al.*, 1960) of sleep (see Fig. 1) and are accompanied by irregularity of heart rate and respiration, and a profound decline of tonus of certain neck muscles in the cat (Jouvet *et al.*, 1960), and the human (Berger, 1961; Oswald, 1962b). The work of Dement and Kleitman (1957b) indicating that dreaming accompanies the REM periods has since been abundantly confirmed. Goodenough *et al.* (1959), however, noted that some persons, when

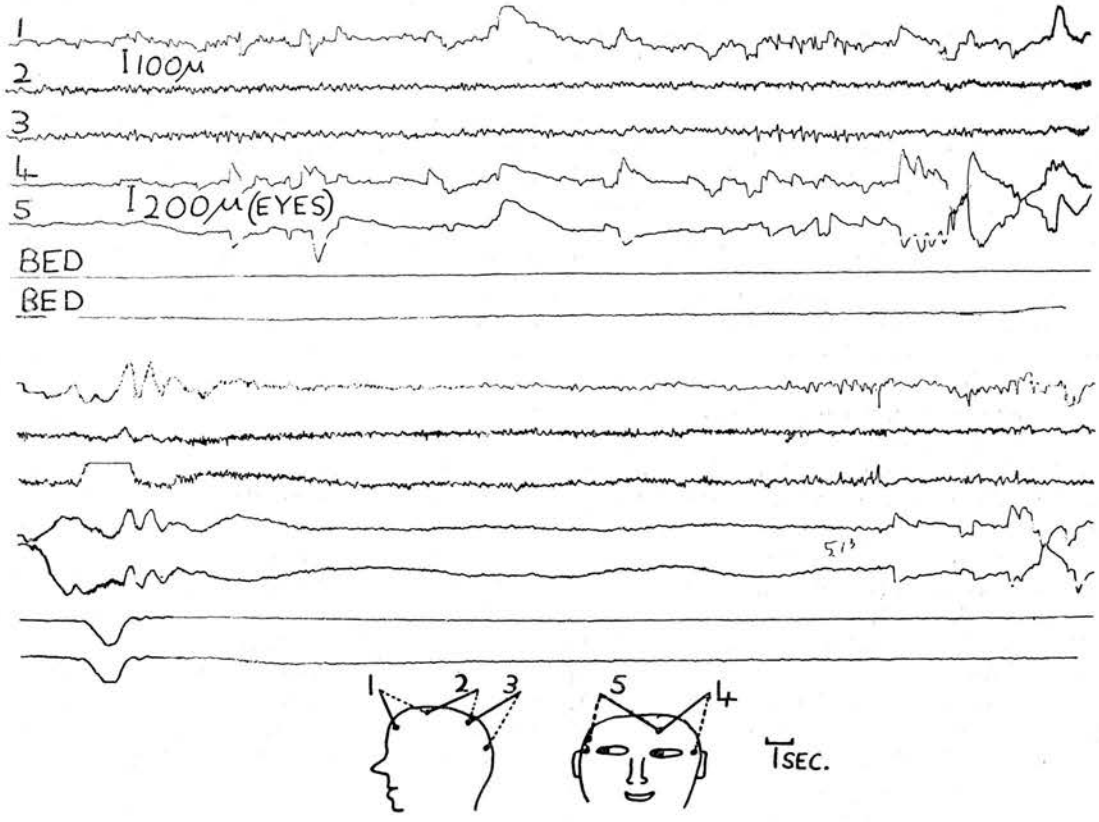


FIG. 1

Control No. 6. A segment of a rapid eye movement (REM) or paradoxical sleep period. The upper and lower excerpts are continuous with one another. Note the rapid, jerky movements of the eyes, followed by more gently rolled eye movements at the time of the major body movement (see deflection of the "BED" channels). EEG rhythms of alpha frequency are associated with the body movement but the slower, low voltage EEG waves of paradoxical sleep then return and rapid eye movements recur, being preceded by a brief burst of "saw-toothed" EEG waves, best seen in leads 1 and 3. Bursts of similar waves can also be seen in the upper excerpt.

awakened from their recurrent REM periods, would not say they had been "dreaming" unless their experiences had been of a bizarre and internally inconsistent nature, but would claim they had been "thinking" and that they had been awake while doing so. The question therefore arose whether patients suffering from autonomous melancholia who claim that they are recurrently awake and "thinking" unhappily during the night might, in fact, be referring to paradoxical sleep rather than to true wakefulness, or whether they might spend an abnormal proportion of the night in such sleep, as the excess of low voltage EEG sleep in such patients claimed by Diaz-Guerrero *et al.* (1946) would suggest.

The cyclical changes in the EEG taken by themselves would suggest that paradoxical sleep is light sleep. Might patients with autonomous melancholia therefore tend to waken, if at all, at these times rather than randomly during the night? If so, they would become selectively deprived of that particular variety of sleep; and such deprivation, when effected deliberately, has been claimed to have adverse psychological sequelae (Dement, 1960).

It was thought probable that such patients do genuinely waken to excess, for they will often

describe rising to make a cup of tea or to contemplate the gas oven. On the other hand, some patients claim to have been awake all night when nurses report them to have been asleep. A nurse might do so, however, while paradoxical sleep is in progress, during which the patient may be conscious of "thinking", and Dement and Kleitman (1957a) have reported that "gross body and limb movements were decreased during rapid eye movement periods" —a fact which has led us to distrust motility measures as indicators of sleep and insomnia (a distrust in no way dispelled by our results). It is, of course, well known that individuals may deny having slept briefly, when the EEG and other physiological variables indicate they have actually slept (Oswald, 1960), and may indeed claim to have been awake all night although the EEG has indicated continuous slumber and they have failed completely to respond to prearranged stimuli (Held *et al.*, 1959; Schwartz and Fischgold, 1960).

Motility studies of the sleep of patients bearing the diagnosis of manic-depressive disease were described by Kleitman (1939). The value of using chronic institutionalized patients is doubtful, however, and we take the view that in these days of the EEG motility studies alone are obsolescent. We therefore carried out motility recording simultaneously with recording of the EEG and of eye movements.

We considered it essential that patients should be compared with normal control subjects studied under identical conditions (alternately with the patients over the course of nearly a year), and that sex and age should be matched. McGhie and Russell (1962) report considerable variations with age and sex of persons' own opinions about their sleep. Since we knew of no comparable controlled study of all-night sleep using barbiturate hypnotics—despite the vast quantities of these drugs in use—we took the opportunity to study the effects of heptabarbitone (Medomin) in clinical dosage.

Diaz-Guerrero *et al.* (1946) carried out an EEG study of the "sleep of patients with manic-depressive psychosis, depressive type". Recordings from six patients on one night each were analysed and comparisons made with analyses published by another author of the sleep of normal subjects. The environmental conditions of sleep, the ages of those studied, and the criteria used in the two groups of analyses may well have varied. One patient slept only 106 minutes, the other five were awake from 5 to 19 per cent. of the possible 8·5 hours. It was reported that the patients spent over twice as much of the night with the low voltage record of light sleep (which we might equate with paradoxical or dreaming sleep) as had been reported for normals. The figures for normals appear to us to be quite remarkably low at 19 per cent. The findings were not supported by any reference to statistical significance, and in view of the small number of nights it is doubtful whether they could have been. The same investigators compared the patients' sleep with that of three control nights specially carried out under similar conditions and reported that the depth of sleep of the controls varied much less frequently during the night, but again no statistical tests were applied and, perhaps more important, no precautions appear to have been taken to minimize subjective bias in the inspection of the recordings. Our investigation was partly designed to test their claims.

Method

Patients and controls each slept for five nights in a bedroom alone. The EEG room was separated from the bedroom by an empty room, but a microphone and amplifier system enabled the subject to be heard if she called out or if she grunted or spoke in her sleep, and even her breathing was audible.

On the first night, intended for adaptation, electrodes were attached to the patient exactly as on other nights, but no actual recording was made. All received two 200 mg. tablets of heptabarbitone on that night. None of the patients or controls had received any hypnotics on previous nights for at least one week, and only patients Nos. 1 and 6 had received hypnotics prior to that. Patient No. 2 had a severe hangover the next morning, to the extent of causing nausea and slight vomiting, and therefore on subsequent nights she received only one tablet, whether dummy or real. Her control, by coincidence,

had an equally severe hangover after the first night and also received only one tablet on subsequent nights.

The second to fifth nights were recording nights. Except as mentioned above, all subjects received two 200 mg. tablets on retiring according to the following scheme on consecutive nights, where D=dummy and M=Medomin.

1. Patient aged 52, control aged 49. M D D M
2. Patient aged 57, control aged 62. M D M D
3. Patient aged 33, control aged 29. D M M D
4. Patient aged 48, control aged 50. D M D M
5. Patient aged 51, control aged 50. D D M M
6. Patient aged 67, control aged 59. M M D D

The dummy tablets were identical in taste and appearance with the real tablets.

An antero-posterior chain of silver cup electrodes containing electrode jelly was attached just to the left of the midline and a further electrode near each outer canthus, enabling the electro-encephalogram and the electro-oculogram to be recorded. Electrodes on the scalp were affixed with collodion and those on the face with adhesive plaster. After a final visit to the toilet, the subject got into bed and the electrode wires were connected to a head box. The wires were collected togther into a bundle with tape and were amply long enough to allow movement. Electrodes attached in this way are comfortable and not noticeable after a few minutes.

A special bed was used. On a divan base was a mattress containing three "Bowden cables" running through its length. One end of each cable was fixed, the other end attached to a steel ruler which it held slightly bent. Any body movement altered the cable tension and the bending force applied to the ruler. On each ruler was a strain gauge. These were in series and any change in the degree to which they were bent altered their electrical resistance and disturbed the balance in a Wheatstone's bridge circuit. When any such disturbance of potential occurred, it could be recorded directly on the EEG machine (see Figs. 1 and 4). It was a highly sensitive device.

All recordings were made with a paper speed of 1·5 cm./sec. and time constants of 0·3 sec. as standard. Recording began when the subject got into bed. This was generally about 10.45 p.m., occasionally slightly earlier or, if technical trouble arose (generally 50 c./sec. artefact), rather later. Recording was continuous through the night except for occasional breakdowns of a matter of minutes (distributed roughly at random through the 48 nights) or when a patient called out for a bed-pan. Recordings ended by 6.30 a.m.

Three EEG channels and two eye-movement channels (see Figs. 1 and 4) were recorded. Two identical channels were used for bed-movement recording in order to detect rare machine-induced brief drifts in one or other channel.

Patients were told that they were to undergo special investigation and that we should gain valuable information about their illness while they slept, which would help us to decide on the best form of treatment. Although they were always asked the next day how they had slept, no stress was laid on this nor any suggestion given that we really wanted to discover how much they slept. They were all given rather more than usual doctor-time by day and they seemed to derive comfort from getting special attention.

The controls were all paid (30s. per night). None had a history of nervous disorder, and except for No. 5 they were all previously known personally to us. Nos. 1 and 6 were domestic staff of Jordanburn Hospital, Nos. 3 and 4 were senior members of the nursing staff, and No. 5 was a blacksmith, the husband of No. 1.

In brief, the clinical features of the patients, which illustrate the diagnostic criteria used, were as follows:

PATIENT NO. 1

Married woman, aged 52. Father had history of mental hospital admissions, dying in last. One sister committed suicide five years before. Another sister treated for depression in another mental hospital two years before. One year feeling depressed, and complaining of insomnia and inability to concentrate. Apathetic, inactive, shunning company. Frequent weeping. At this stage, used in present investigation.

Six E.C.T. with recovery. Discharged three weeks later. After two weeks, admitted under certificate to another mental hospital on account of acute mania. Recurrent manic and hypomanic episodes for six months, finally discharged after further three months.

PATIENT No. 2

Married woman, aged 57. Mother an energetic midwife. Five miscarriages, no children. Mildly depressed at menopause 1946. Typical depressive illness 1957, treated successfully with E.C.T. Also 1959, when again had E.C.T. Very active, bustling woman when well. Pyknic build. November 1960, had 'flu, depressed after. Seen in March 1961. Sitting about in agitated state, weeping, unable to do housework for month. No interest, "tired", waking in the night and weeping, shunning company. Wanting to "sleep away" from life. Speaking little except repeatedly about having had no children.

Seven E.C.T. with recovery. Follow-up after three months revealed a lively, ebulliently cheerful woman with no complaints; very busy.

PATIENT No. 3

Married woman, aged 33. Mother died in childbirth. One sister had puerperal depressive illness. During fifth (unwanted) pregnancy, became depressed. Onset of severe agitated preoccupation two weeks after birth. Thoughts that she should kill her children, husband and self, by gassing. Self-reproach great. Fears of bodily disease and insanity. No interest. Complaint of broken sleep. Much weeping. No previous psychiatric trouble. Used in this investigation.

Nine E.C.T. Discharged cheerful, well and active. Relapsed after month. Imipramine, 150 mgms. per day, as O.P. Gradual recovery, discharged five months later, advised imipramine continue six months. Unfortunately, it was stopped. Relapsed. Seen two months later, extremely depressed, retarded, unkempt, sitting weeping, not eating, complaint of insomnia, preoccupation with death and suicide. Five E.C.T. and imipramine, 150 mgms. per day, rapid improvement but not fully recovered for seven months.

PATIENT No. 4

Married man, aged 48. Printer's assistant. Parents' marriage broke up when patient aged 12. Over-strong attachment to mother, visiting her daily even after own marriage. Past history of haematemesis, perforated duodenal ulcer and partial gastrectomy 13 years ago. Grief reaction to mother's death seven months before had changed into state of increasing apathy, loss of interest in normal activities, inability to work for a week, anorexia and loss of weight, inability to concentrate, fears of insanity. "Thoughts racing" while lying awake at night, suicidal ruminations and misery. Frequently overcome by strange fears (of crossing the road, etc.); avoided company; feeling people might be talking about him. Complaints of bodily weakness; of weight pressing on head; fears of serious chest disease. His wife and workmates found him silent and tearful, quite unlike his usual "life and soul of the party" way.

Six E.C.T. Became temporarily overactive. Back to work two months after first attendance. Discharged a month later—"champion in my spirits".

PATIENT No. 5

Married man, aged 51. Miner (underground). Rigid upbringing in narrow religious sect. Attended August 1961 after feeling tired and unwell for a year. Tonsillectomy November 1960 had not revived him. Off work two months; total loss of interest, sitting staring out of window all day, trembling; fears of "collapsing". Often near to tears. Claimed not slept at all for several weeks; band-like discomfort round head. Never like it before in life. Enquiries at work revealed a well-liked, conscientious worker.

Nine E.C.T.; greatly improved; a few complaints about his head. After four weeks, feeling "numb" in head, fears of "clot of blood", feelings of hopelessness. Imipramine, 150 mgms. per day, started, with steady improvement. December 1961, feeling very well, lively, cheerful, back at work underground.

PATIENT No. 6

Woman, aged 67, single. Father, an energetic man, committed suicide by drowning, aged 69. No previous personal psychiatric trouble. Became agitated and miserable January 1960. On day before admission, April 1960, made serious suicidal attempt (coal gas). After resuscitation, treated with nialamide with moderate improvement. Worsening of depression, readmission September 1960, recovery after eight E.C.T. Well until July 1961. Readmitted October 1961. Miserable, weeping, "throbbing" in head, sternum and stomach, "weakness" in legs, everything an effort, shunning company, wishing could die. Reported insomnia, and improvement of mood in later part of day. Used in this investigation.

Treated with hypnotics and with imipramine, 75 mgms. per day. Steady recovery. Six months follow-up; very well, cheerful, busy, sewing, reading, playing piano, singing at her housework; going out each afternoon.

ANALYSIS OF RESULTS

Since all EEG interpretation is subjective, and since it was necessary to determine the length of time per night in the various stages of sleep, it was important to minimize subjective bias. Therefore, the 48 recordings were eventually each assigned a code number in irregular order (by R.J.B.) and each was presented, again in irregular order, to one of us (I.O.), who was therefore not aware whether the recording was from a patient or from a control. Heptabarbitone does not seem to produce much barbiturate fast activity in the EEG and, in fact, the scorer was never aware whether a particular record was of a night on which real or dummy tablets had been given.

A page by page analysis was made; the time of first falling asleep (onset of spindles or

Stage C) was noted, and the times of all subsequent changes of sleep depth, including very brief ones following a movement in sleep, provided the change persisted for 10 seconds or more. The time of each movement was noted and whether it was isolated or part of a quick succession of movements (so that, when desired, the latter could be scored as one major movement in some of the statistical analyses). It must be pointed out that each movement was seen as a brief oscillation and no account was taken of its size or of the number of changes of pen-direction within what clearly represented a single movement of the body. The movement scores therefore differ in nature from those of some other workers who have recorded some integrated form of total movement energy output. The time of the first and last REM in any unbroken part or the whole of a REM period was noted.

The stages of sleep noted were those described as A to E by Loomis *et al.* (1937). Stage A corresponds to wakefulness or near-wakefulness; included as Stage A were brief periods of 10–120 seconds where rhythms of alpha frequency covered most of the head after a movement and arousal from, say, Stage C. There are many such brief periods in nocturnal sleep and it is very doubtful whether the disoriented slumberer becomes sufficiently aware of reality at these times to remember them in the morning. A few muttered, incoherent words may accompany the initial movement (more rarely a few words are muttered during the REM periods). Stage B denotes a low voltage record with predominant 3–6 c./sec. waves. An EEG pattern of this nature appears when the individual passes from wakefulness to Stage C, but also during the REM periods. The EEG characteristically found during the REM periods has been designated Stage B in this investigation and accounts for all but a very small amount of the Stage B scores. It often begins just before, and usually persists longer than the REMs and (Dement and Kleitman, 1957a) is a better measure of dreaming-time than the "REM times" in the scores—namely time from first to last REM within any unbroken period of Stage B with REMs.

Actually, the unique saw-toothed EEG waves mentioned previously were commonly the first sign that a change was taking place from Stage C to Stage B with REMs, frequently appearing as a brief burst half a minute before the change from Stage C to Stage B.

When all the records had been thus analysed, the code was broken (by R.J.B.) and statistical analyses made, using analysis of variance and a two-way classification: patients/controls and dummy/Medomin.

The times at which the subjects first fell asleep (onset of spindles or Stage C) varied from 10.23 p.m. to 12.17 a.m., with the exception of Patient No. 1, who on her second dummy-tablet night did not fall asleep until 3.41 a.m.: for that night, the arbitrary time of 12.00 midnight was taken as the time of commencement for data included in the statistical analysis.

Recording generally ceased between 6.00 and 6.30 a.m. However, in a few instances, patients terminated the recording earlier by getting out of bed, switching on the light, etc., following a period of wakefulness. Only in the case of the second night of Patient No. 1 did this occur earlier than 5.30 a.m. No arbitrary correction in respect of these few variations has been made to the figures for the number of movements and the time in Stage A, the figures for which would presumably have been increased had the patients continued to lie awake in bed and recording been continued. Equally, no corrections have been made for the fact that a few minutes were occasionally lost through, for instance, paper-stoppage—such incidents were presumed to be random.

An attempt was made to determine whether heptabarbitone affected the amount of rapid eye movements occurring within a given duration of a REM period. The records of the controls only were taken and each of their heptabarbitone night records was examined by R.J.B. so that periods of five minutes at the start of the second and third REM periods of the night were chosen. In two instances, only one REM period of sufficient duration was present.

In the case of each recording, the corresponding duration within the corresponding REM period of the recording on the dummy tablets was also chosen. The relevant portions of the records were then all presented in random

order and with only a code number to I.O., who counted what he judged to be the individual rapid shifts of eye-direction. The code was then broken and the results analysed.

Results and Comments

The main results are summarized in Table I, which shows mean values. Where probabilities are not shown in the three right hand columns, they did not approach statistical significance.

A. *Whole night*

The mean total recording time spent per night in Stage A, or wakefulness, after the time of first falling asleep, was significantly greater ($P < \cdot 01$) in the patients than in the controls, and was significantly reduced by the drug ($P < \cdot 01$).

The figures for the total time spent in each of the Stages B, C, D and E of sleep are expressed as mean percentage of total time spent asleep on each night, rather than as absolute figures of mean total duration spent in each stage. Differences in percentage figures thus indicate differences in the overall sleep pattern, whereas differences between absolute figures might result from differences in total sleep time owing to different times of falling asleep and variation in time later spent awake and in hour of rising. For the same reasons, movements and shifts of depth of sleep are expressed as mean frequencies.

Heptabarbitone significantly reduced the frequency of movement ($P < \cdot 05$), and shifts of depth of sleep, both from one stage to another and also to Stage A alone ($P < \cdot 025$). Most striking was the reduction of percentage time in Stage B sleep ($P < \cdot 001$). Most of this time was contributed by the REM periods and the EEG Stage B which accompanies and often exceeds the duration of REMs. The percentage time of the night occupied by actual REM periods was similarly reduced by the drug ($P < \cdot 001$).

The percentage time spent in Stage C sleep by patients was significantly less than controls ($P < \cdot 025$), but the percentage time spent in Stage E sleep significantly greater ($P < \cdot 05$). These figures suggest that patients, since they spend significantly more time awake at night, are sleep deprived so that when they do sleep they spend more time in Stage E sleep (traditionally identified as deep sleep), for the latter is known to be increased after sleep deprivation (Berger and Oswald, 1962a). The finding might also be influenced by the fact that, as the number of hours asleep per night increases, there is normally a decline of time in Stage E and increase in time of Stage C.

As previously noted, patients were not pressed to describe how they had slept, but the descriptions they did volunteer showed a much closer agreement with that which the EEG revealed than had been expected.

B. *The hours* 1.30–5.30 *a.m.*

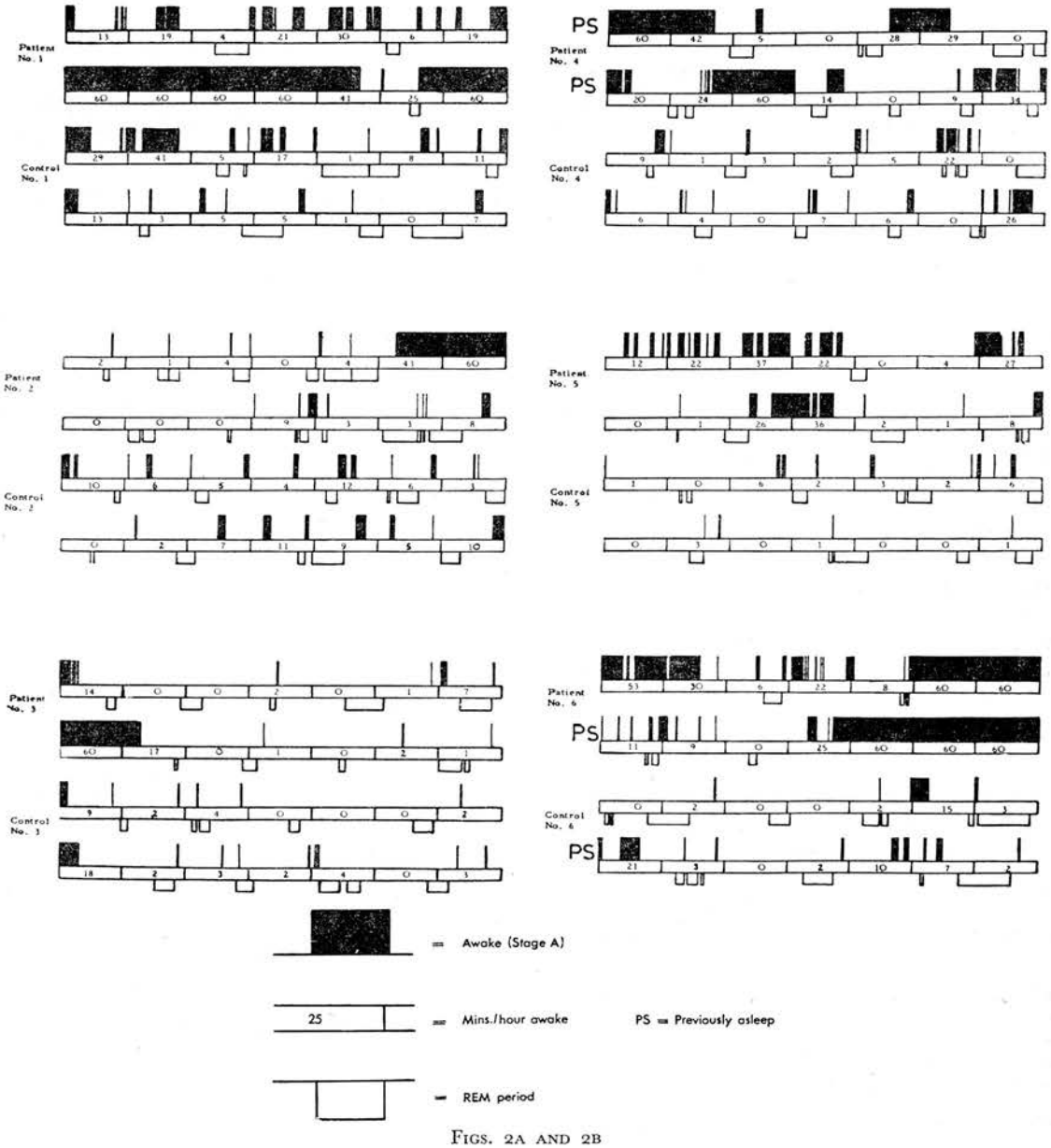
At the outset of the study, it was decided that particular attention would be paid to the time 1.30–5.30 a.m., the early morning hours during which the patient suffering from melancholia traditionally lies awake. During these hours, outside noises from vehicles and hospital activities are minimal, and the figures are independent of variations in time of getting to sleep, some of which arose from technical difficulties in applying electrodes and getting artefact-free recordings.

Patients spent very much more time awake during these hours ($P < \cdot 01$). Heptabarbitone greatly decreased time awake ($P < \cdot 001$), especially in the patients (there being a significant interaction, $P < \cdot 025$) in whom, of course, there was much more scope for its effect.

A significant interaction between the two variables was present in the mean time spent in Stage B sleep. Patients on dummy tablets spent significantly less time in Stage B than controls, but this can be accounted for by the diminished total time spent asleep during those hours among the patients. Similarly, patients spent significantly less time in Stage C ($P < \cdot 01$). Heptabarbitone increased ($P < \cdot 001$) time spent in Stage C, at the expense of time awake and Stage B.

C. *The pattern of wakefulness*

It had been an object of special interest to determine whether the periods of wakefulness among the patients had a relation in time to the cyclical changes in the character of sleep during the night. The numerical findings do not give any indication of, for instance, selective wake-



FIGS. 2A AND 2B

Sleep pattern between 11 p.m. and 6 a.m. on the 24 *dummy* tablet nights of both patients and controls. Each transverse block system represents one whole night.

The periods of wakefulness of EEG Stage A have been plotted except where the duration of such episodes was less than one minute. The greater proportion of the night spent awake by the patients compared with the controls can be readily seen, the number of minutes awake in each individual hour between 11 p.m. and 6 a.m. being shown. The lower, unfilled blocks represent the time between onset and offset of unbroken REM periods, from first to last rapid eye movement. No obvious relation between REM periods and wakefulness in the patients can be seen. Even when sleep is much reduced some of it is taken as paradoxical sleep with REMs (see, for instance, the second nights of patients No. 1 and No. 4).

fulness in place of REM periods. It was necessary, however, to examine the distribution of wakefulness, which may be seen in Fig. 2, in which the times of awakening for periods of one minute or longer appear to be random, and in which it may be seen that even when little sleep was obtained a proportion was occupied by REM periods.

D. *Body movements*

Separate analyses were made in respect of scores of individual movements and of scores where a closely grouped series of movements was counted as a single movement. The latter manoeuvre did not reveal any additional information, and only data of the former class are given in Table I.

As previously noted, other workers have attempted to study the sleep of psychiatric patients using motility indices, but when these were used concurrently with the EEG we found the latter a very much more sensitive discriminator between both classes of variable. In fact, mean total movements over the whole night and between 1.30 and 5.30 a.m. (see Table I) failed to discriminate at satisfactory levels of statistical significance. In our recordings, we observed, as did Brooks *et al.* (1956) and Coleman *et al.* (1959), that, in general, movements were fewest in the earlier hours of the night, when the high voltage EEG Stages D and E were most manifest. It is, however, worth emphasizing that there are big individual differences. Some people will move much more in the first half of the night than the second (Fig. 3) even while sleep is, by traditional EEG criteria, deep, with the Stage D pattern present (Fig. 4); others will lie awake and move little.

A comparison was made, based on dummy tablet nights only, of the frequency of movements in sleep of D and E Stages on the one hand and in paradoxical sleep with Stage B EEG in association with REM periods, on the other.

Two different methods of assessment were made, namely, D + E "pure" and B "pure"; D + E "overall" and B "overall". The "pure" scores were derived from the number of movements during discrete periods of the Stage B record or the record of Stages D and E. The

TABLE I

	CONTROLS		PATIENTS		P VALUES OF STATISTICAL SIGNIFICANCE (Analysis of Variance)		
	Dummy	Medomin	Dummy	Medomin	Medomin/ Dummy	Controls/ Patients	Interaction
WHOLE NIGHT							
Movements	77	70	117	63			
Movements/min. × 100 ..	18	16	28	5	·05		
% Time A Stage (min.) ..	45	24	109	37	·01	·01	
% Time B Stage	28·4	17·8	25·6	19·6	·001		
% Time C Stage	45·0	55·1	39·0	41·9		·025	
% Time D Stage	20·3	22·8	27·4	27·6			
% Time E Stage	5·6	4·4	8·0	11·2		·05	
% REM Time	23·3	11·8	20·6	14·8	·001		
Shifts of depth/min. asleep × 100	19	17	25	18	·025		
Ditto to A Stage	6	5	9	5	·025		
1.30 TO 5.30 A.M.							
Movements	40	35	69	40	·06	·06	
Time A Stage (min.) ..	23	12	76	18	·001	·01	·025
Time B Stage (min.) ..	68	49	48	54			·05
Time C Stage (min.) ..	104	134	64	108	·001	·01	
Time D Stage (min.) ..	37	36	38	49			
Time E Stage (min.) ..	3·8	4·1	4·8	10·3			

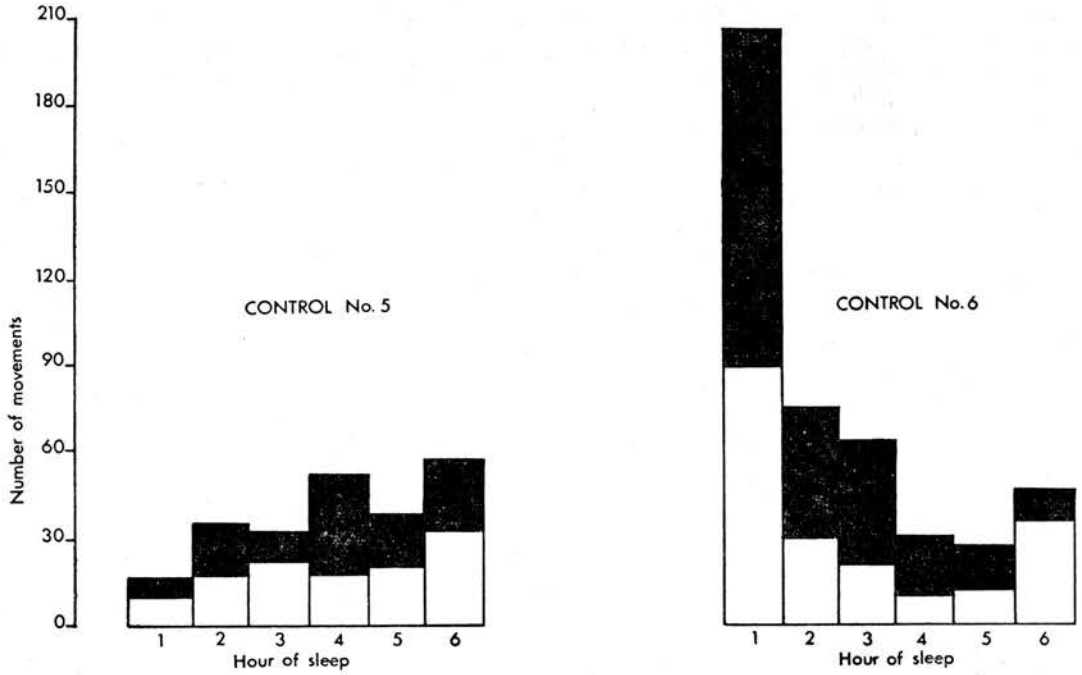


FIG. 3

The histograms show the number of movements per hour for the first 6 hours after first falling asleep. The solid areas indicate the sum of the movements during the two heptabarbitone nights, plain areas indicating dummy tablet nights.

The contrast between the distribution of movements within the nights of these two subjects should be noted. Although most of the movements by No. 6 occurred early in the night they took place actually during sleep, for example, only 19 movements in the first hour and 5 in the second hour occurred during brief periods of wakefulness.

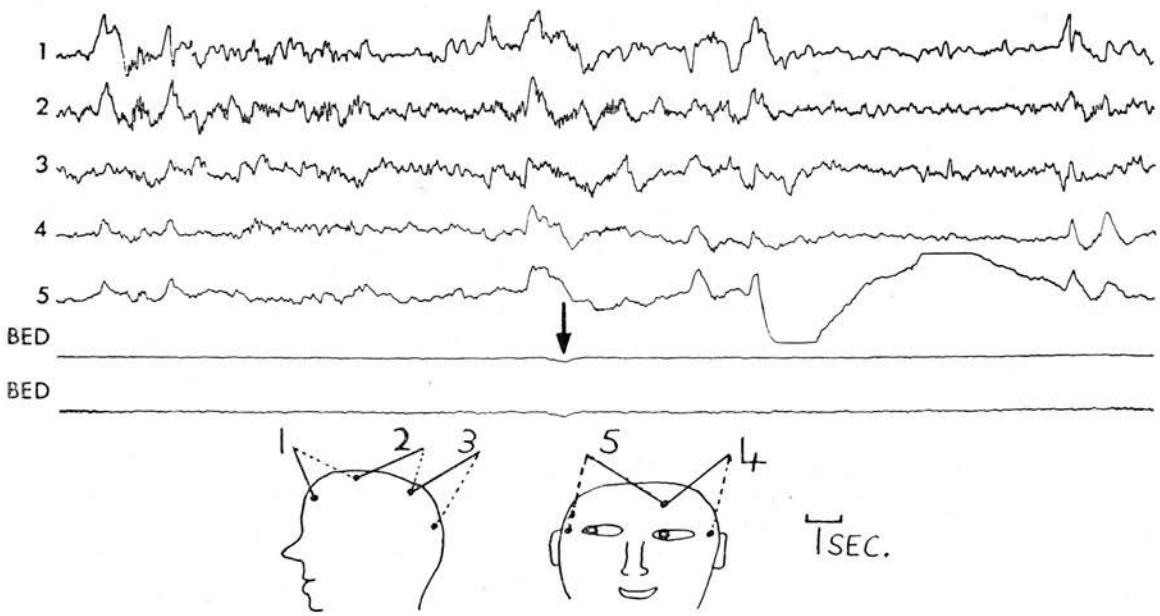


FIG. 4

Control No. 6. The EEG shows the high voltage slow EEG waves and the faster spindles of Stage D (contrast with Fig. 1). The EEG potentials are picked up by the electro-oculogram (channels 4 and 5) but actual eye movement potentials are absent. Despite the deep sleep the BED channels reveal one of many body movements (see Fig. 3).

"overall" scores were obtained by noting the number of movements between the first and last moment of Stage B or Stages D and E respectively within any given cycle (by EEG criteria, sleep appears to undergo cyclical rises and falls of depth during the night) even if there were gaps of several minutes with Stage A or Stage C intervening. A movement often terminates a period of both Stage B and Stage D or E, and these terminating movements were included in the scores (only the first movement if there were a group of them).

Mean "pure" movements × 1000/min. with EEG Stage B

in patients=146
in controls=168

Mean "pure" movements × 1000/min. with EEG Stages D and E

in patients= 62
in controls= 79

Mean "overall" movements × 1000/min. with EEG Stage B

in patients=138
in controls=186

Mean "overall" movements × 1000/min. with EEG Stages D and E

in patients=101
in controls= 97

Analysis of variance showed no significant difference between controls and patients, but the much higher frequency of movement in Stage B than in Stages D and E was significant ($P < \cdot 001$ in the case of the "pure" category, and $P < \cdot 05$ in the case of the "overall" category).

A further assessment was made of movements during discrete periods of Stage C record (or C "pure").

Mean movements × 1000/min. with EEG Stage C

in patients=137
in controls=139

Analysis of variance showed that though the figures were less than those for Stage B the difference was not significant. On the other hand, the frequency of movements in Stage C was significantly ($P < \cdot 025$) greater than in Stages D + E ("pure").

E. *The frequency of rapid eye movements*

The actual frequency of rapid movements of the eyes *during* a REM period in the controls was diminished in every case by the heptabarbitone except in the case of control No. 2 (who, it will be recalled, received only half as large a dose). The analysis covered a total of 3 hours 40 minutes and the actual scores of eye movements for total individual samples of 20 minutes (except for controls Nos. 4 and 5, where the samples totalled 15 minutes) were as follows:

		Dummy	Medomin
Control No.	1	831	311
	2	238	267
	3	577	326
	4	300	254
	5	262	235
	6	899	481

A t-test for significance of mean difference between the paired 5-minute samples showed that this reduction of the frequency of eye movement activity within on-going REM periods was significant ($P < \cdot 02$).

Discussion

A notable feature of the investigation was its failure to reveal any characteristic abnormality of sleep in melancholia other than the excess of wakefulness. Wakefulness recurred frequently during the night rather than sound sleep followed by early final wakening (Fig. 2). In particular the episodes of wakefulness were not related to the recurrence of paradoxical sleep or REM periods. Furthermore, the claims of Diaz-Guerrero *et al.* (1946) were not substantiated; patients showed no excess of light sleep, in fact the reverse, while they did sleep; and their slightly greater frequency of shifting of sleep depth was not statistically significant, and bore no similarity to the large difference in this respect claimed by Diaz-Guerrero and his colleagues from their much smaller series.

Apart from the expected increased duration of sleep, the outstanding effect of heptabarbitone was the reduction in paradoxical sleep—a reduction of dreaming. This is of interest in view of the evidence that there is a

specific need for this kind of sleep, deprivation of which may have adverse sequelae (Dement, 1960). We do not of course know whether the effect might disappear if heptabarbitone were repeated over a larger series of nights.

The reduction of body motility during the night caused by heptabarbitone is consistent with the similar reduction by pentabarbitone (Brazier and Beecher, 1952; Hinton and Marley, 1959).

It has been claimed that the rapid conjugate movements of the eyes represent scanning movements in response to the visual imagery of the dream (Dement and Wolpert, 1958). In conformity with such a view is the absence of REMs in those with life-long or nearly life-long blindness (Berger *et al.*, 1962). The alternative is simply to regard the REMs and the associated saw-toothed EEG waves as neurophysiological concomitants of paradoxical sleep without reference to conscious content. The fact that heptabarbitone not only decreases paradoxical sleep duration but also depresses one of its most striking features, namely the frequency of eye movements, could either be interpreted as a demonstration of a simple neurophysiological effect or it could be argued that barbiturates have a tranquillizing effect on fantasy life, so that there is less activity at which to "look" during dreaming—thus it has been claimed that dreams of, e.g., television viewing, are accompanied by less REM activity than, say, watching a lawn tennis game (Dement and Wolpert, 1958; Berger and Oswald, 1962b).

Aserinsky and Kleitman (1955) first reported increased body motility in association with REM periods, but then Dement and Kleitman (1957a) described decreased motility during REM periods. Furthermore, some authors have felt inclined to describe paradoxical sleep as "deep" sleep (e.g., Rossi *et al.*, 1961) owing to the raised threshold for full arousal of the cat, on reticular formation electrical stimulation (Benoit and Bloch, 1960).

It is traditional to use the adjectives "light" and "deep" in respect of sleep, the degree of inertia and unresponsiveness being greater in the latter. The EEG during paradoxical sleep is, apart from the saw-toothed waves, of the kind traditionally identified with light sleep, and indeed the presence of consciousness, even if only of a fantasy life, at these times is consistent with such a view. One of us had in the past taken the view that paradoxical sleep was light sleep (Oswald, 1962a) but, because of the muscle tonus changes previously mentioned, has now to accept that the term "light" sleep is inappropriate and that paradoxical sleep is a *different kind of sleep*. Nevertheless, in so far as one would use the adjective "deep" to describe sleep associated with few major body movements, when a precise determination is made, as in section D of our results, it is clear that the adjective "deep" is more appropriate to human sleep of EEG Stages D and E than to human paradoxical sleep; significantly more movements occurring in association with the latter. Furthermore, the frequency of movements in Stage C sleep, being intermediate between the other two categories, was consistent with the traditional classification of sleep depth in terms of the EEG.

Summary

Continuous nocturnal recording of EEG, eye movements and bed-movements was carried out on six patients, not previously receiving hypnotics, and six sex and age-matched controls. They were involved on five successive nights each—the first night being ignored. The patients were ill with typical melancholia ("endogenous depression" or "manic depressive psychosis/depressive type", 301·1 in the International Classification). According to a planned design, each patient received 400 mg. of heptabarbitone (Medomin) on two nights and dummy tablets on two nights.

All the recordings were analysed by one of us in ignorance of whether the record was that of a patient or of a control. Findings included: (1) Patients spent significantly more of the night awake, although times of awakening were not related to the recurrence of rapid eye movement periods ("paradoxical sleep"). Percentage time spent during the latter, and frequency of shifts in depth of sleep, did not differ significantly from the controls. (2) Heptabarbitone greatly decreased duration of rapid eye movement (dreaming) periods and also the frequency of eye movements within those periods. (3) Heptabarbitone decreased time awake, especially in patients in early hours of the morning,

and decreased frequency of shifts of sleep depth and frequency of body movement. (4) The EEG discriminated far better than body motility, which is subject to large individual variations. (5) Body motility decreased according to traditional EEG stages of sleep depth, being significantly greater in association with "paradoxical sleep" than in association with traditional "deep" sleep.

Acknowledgments

The work was conducted in the Professorial Unit, Royal Edinburgh Hospital. We wish to acknowledge the encouragement of Professor G. M. Carstairs and Professor J. Drever, and we should like specially to mention Dr. F. J. Fish, to whom we are indebted for his teaching about depressive illness. The research project was made possible by the generosity of Messrs. Geigy Pharmaceutical Company, Ltd., and of Messrs. Slumberland (Research) Ltd. A grant towards the cost of EEG paper was received from the University of Edinburgh Moray Endowment Research Fund. One of us (R.J.B.) was in receipt of a research grant from the Alexander Kennedy Research Fund and subsequently of a grant from the Medical Research Council. One of us (R.A.J.) was in receipt of a Rockefeller Fellowship. We should like also to express our thanks for statistical advice given us by Dr. D. N. Lawley of the Department of Statistics, University of Edinburgh.

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ACCEPTED FOR PUBLICATION IN THE BRITISH MEDICAL JOURNAL.

(18)

AMPHETAMINE AND PHENMETRAZINE ADDICTION: PHYSIOLOGICAL ABNORMALITIES IN THE ABSTINENCE SYNDROME.

by

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Amphetamine addiction is common, though the readiness with which such addiction develops is not as widely recognised as one could wish. Those addicted frequently take large quantities of short-acting barbiturates as well and, of the tablets containing both, "drinamyl" (dexamphetamine sulphate 5 mgms. with amylobarbitone, 32 mgms.), popularly known as "purple hearts", are in most common use. A popular substitute or equivalent for amphetamine is phenmetrazine ("preludin"). . . "known in London as 'sweeties' . . available in any quantity . . at the rate of £1 for 24 tablets" (Kellock, 1962). This compares with the price of £1 for 25 tablets amongst Edinburgh factory-workers (Case 4 below). Experience of addiction to these drugs (Bell and Trethowan, 1961) in New South Wales led Trethowan (1962) to emphasise the progressive character deterioration associated with such addiction. He also pointed out that to claim, as many do, that they are drugs, not of addiction but only of "habituation", is to engage in a sematic quibble.

The Interdepartmental Committee (1961) appointed by the Ministry of Health and the Department of Health for Scotland, though remarking that in 1959 there were no less than 5,600,000 prescriptions for preparations of the amphetamine and phenmetrazine, "formed the impression that. . . abuse is not widespread". The Committee drew a distinction between addiction and habituation, partly on the grounds of "physical dependence on the effects of a drug" in the case of addiction and "absence of physical dependence and/

and hence of an abstinence syndrome" in the case of habituation.

Addiction to these drugs is common in Edinburgh, and it is our purpose to demonstrate that physical dependence and characteristic, persisting and easily measurable "physical" or physiological abnormalities form part of the abstinence syndrome.

Two Varieties of Sleep.

Amphetamines have been much used for preventing sleep, and have been shown to act upon the reticular formation (Hiebel et al., 1954). In recent years, there have been considerable advances in knowledge about sleep, and it has now to be recognised that there are two principal kinds of sleep (Oswald, 1962; 1963) which have been called the "fore-brain" and "hind-brain" (or "paradoxical") phases of sleep respectively by Jouvet (1962). Human "fore-brain sleep" is accompanied by high voltage slow waves and spindles in the electroencephalogram or EEG, ocular repose and incomplete relaxation of skeletal muscle. Human "hind-brain sleep" is accompanied by bursts of conjugate rapid eye movements (REMs) and an EEG of fairly low voltage (Fig. 1) somewhat similar to that of drowsiness but with periods of characteristic "saw-toothed" waves of 2-3 c/sec. "Hind-brain sleep" is accompanied by diminution of remaining muscle tone in the cat (Jouvet, 1962) and in the human (Berger, 1961). When volunteers are awakened from this kind of sleep, they report having just been dreaming (e.g. Dement and Kleitman, 1957a; Berger and Oswald, 1962b).

Human/

Human "hind-brain sleep" does not appear except following prior "fore-brain sleep". It recurs cyclically, occupying 4-6 discrete periods of the night. The transition from "fore-brain" to "hind-brain" sleep is abrupt. Though the reverse transition is usually also abrupt, sometimes it can only be determined to within the nearest couple of minutes. In order to obtain a sharply determinable measure of duration, one may note the "REM-time" by recording the duration of the period from the first to the last rapid eye movement in any period of "hind-brain sleep", subtracting the duration of any interruptions (generally following a major movement, when the EEG may briefly become that of "fore-brain sleep" with "spindles" or that of wakefulness) and determining the sum for the whole night. This may then be expressed as a percentage of the total sleep time.

In man, the first episode of "hind-brain sleep" occurs about 1-1½ hours after falling asleep, lasts a few minutes, then ceases. In about a quarter of instances, and especially in some individuals, no actual REMs occur during the first episode though they do in later episodes during the night.

It seems we need a fairly fixed proportion of each kind of sleep, experimental deprivation of "hind-brain sleep" being followed by a compensatory increase when opportunity allows (e.g. Dement, 1960; Berger and Oswald, 1962a), though never approaching the values reported below. Retrospective examination of records from a previous study (Oswald et al., 1963) in which it was shown that barbiturates reduce the REM-time, also suggests a compensatory increase early in succeeding nights when the drug was not administered.

Physiological Abnormalities on Amphetamine and Phenmetrazine Withdrawal.

Six addicts admitted for drug withdrawal between June and November 1962 were studied. It is difficult to persuade such patients to enter hospital and if one stops their drugs abruptly they may discharge themselves. They were therefore allowed to continue their drugs briefly in order to allow a doctor-patient bond to develop, strong enough to sustain them on drug withdrawal. Their nocturnal sleep was studied, either by whole night recordings of the EEG and eye-movements, and sometimes muscle tonus, or, in a few instances, by recordings of the first 2 hours of sleep only. The first appearance of "sleep spindles" at about 14 c/sec. in the EEG was taken as the time of sleep onset.

Silver disc electrodes containing electrode jelly were fixed to the face near the outer canthi with adhesive plaster, in order to record eye movement potentials, and to the scalp with collodion in order to record the EEG (Fig. 1). Electrodes thus fixed are not noticeable after the first few minutes. A paper-speed of 1.5 cm./sec. and time constants of 0.3 sec. were used. Patients slept in a bedroom away from the EEG room. In all, 88 recordings, running over some 24 miles of paper and of which 83 were whole-night recordings, were made on 60 nights. Sometimes we recorded from two patients concurrently. Recordings generally began around 2300 hours and ceased around 0730 hours, depending on when the patient woke spontaneously or was awakened by outside noises or by the patient sharing the room.

Case/

Case 1. A 41-year old woman. Given, from admission on 1st June, her usual 10 drinamyl daily. First sleep recording on 5th June. No drinamyl on 14th, 15th, 25th and 26th June, and it was finally stopped on 5th July.

Withdrawal (Fig. 2) was followed on each occasion by a dramatic rise of REM-time to values far exceeding those reported or encountered previously.

Case 2. Married woman of 20. Admitted 15th August, 4 tablets of durophet (50 mgms. of amphetamine) permitted daily. Durophet stopped 20th August. Fig. 3 shows the same kind of withdrawal effect as was seen in the other patients.

Case 3. A 40-year old woman. Permitted "drinamyl", 15 tablets each morning, till stopped on 18th July. On 20th, dexamphetamine sulphate, 50 mgms. each morning, begun. This stopped on 9th August, and on 11th August only 30 mgms. daily was resumed (comparable to doses often prescribed on "therapeutic grounds"). On 20th August, 45 days after admission, drugs ceased. Figs. 4 and 7 show the effect of drug withdrawal.

On 22nd September, she was in unusually good spirits, so her urine was examined for amphetamines, but no trace of these was found. The examination was kindly carried out by Dr. J. D. Crombie, and involved extraction, vacuum concentration and one-way paper chromatography in a butanol-acetic acid water system, the spots being visualised with diazotized p-nitro-aniline reagent. This method has proved a sensitive qualitative detector with other such patients.

Case/

Case 4. A 50-year old married woman of below average intelligence.

Admitted September 2nd; permitted eight 25 mgms. tablets of preludin (phenmetrazine hydrochloride) until 8th October. In the second week after stopping her drug, two surprise urine checks were made. Chromatography revealed no trace of phenmetrazine.

Fig. 6 shows an effect of phenmetrazine withdrawal similar to that of amphetamine withdrawal, recovery to her probable normal levels of function taking place in 3-4 weeks.

Case 5. A 39-year old married woman. Admitted 29th August; allowed 9 tablets of "drinamyl" each morning until September 16th.

Figs. 5 and 7 illustrate the slow return to normal sleep function after withdrawal.

Case 6. A 25-year old unmarried woman. Admitted 6th November. Allowed 9 preludin daily till 10th November. Her sleep was recorded on 4 nights only. On the nights of 6th and 7th November, she had no REMs within the first 2 hours on either night, but after withdrawal 29 and 26 min. on the 10th and 12th respectively. The whole night percentage rose from 15.5 (42 min, in 270 - Case 4 wakened her early) and 24.3 (112 min. in 461) to 31.7 (170 min. in 536) and 27.5 (123 min. in 448). The delay before the first REMs fell from 123 and 132 min. to the unprecedentedly low figure of 4 min. on each night.

The/

The fully-addicted patients slept remarkably well despite their stimulant drugs. The delay between onset of sleep and the first REM period tended to fall considerably on some of the first nights after drug withdrawal, thereafter slowly rising (Table I). After withdrawal, patients tended to fall asleep sooner after retiring and the duration of sleep during the night tended to rise, subsequently to fall again (Table I).

Cases 1, 3, 5 and 6 all had some extremely short delays on drug-withdrawal. We followed two over a long period, until three consecutive delays within normal limits (Fig. 7). At times, these delays were lengthened by the patient first falling asleep, wakening after a few minutes, and so on several times. In addition, when the patient wakens for, say, 20 minutes just as the first REM period is expected, then REMs do not appear till the second cycle of the night is due - Case 3, August 20th, and Case 5, October 17th, delays were lengthened thus.

While patients were receiving the drug to which they were addicted, the variables we have measured mostly fell within normal limits. Patients received their drugs in the mornings only, and by nightfall a minor degree of deprivation may have contributed to the three very short delays (Cases 1, 3 and 4, Table I), and to the REM time of Case 1 on 19th June (whole night) and of Case 3 on 18th August (first 2 hours).

Discussion.

To establish the existence of "physical" dependence on a drug, it is necessary to demonstrate some physiological function which/

which is within normal limits in the fully addicted patient but which becomes abnormal upon drug withdrawal, and which returns to normal if the drug is again given. The physiological function we have chosen to measure concerns a basic brain process - sleep, upon the mechanisms of which amphetamine was known to have an action. Fisher and Dement (1962) and Maron and Rechtschaffen (personal communication) have observed that the decrease in whole-night REM-time caused by a barbiturate can be significantly enhanced by giving amphetamine with the same quantity of barbiturate. The central nervous systems of our patients had evidently become so accustomed to amphetamine that a normal pattern of sleep was present. In each case (Figs. 2-6) upon withdrawal a huge increase in REM-time occurred, reversible by reinstituting the drug (Figs. 2 and 4). Alternatively, the passage of time brought about decline to fairly steady individual, normal values. The addicts were therefore dependent on their drugs for normal function.

Each individual has his own mean and usual range of REM-time values. As a guide, we have included in the figures an indication of the upper limits of normal for the population as a whole in so far as they are currently available - from Dement and Kleitman (1957b), Dement (1960), Berger and Oswald (1962), other unpublished studies in our own laboratory, and Rechtschaffen and Verdone (1963) - in the sense that the following figures are unlikely to be exceeded more than once or twice per hundred instances, namely, 32% REM-time in the whole night (mean about 20-22%); and 30 minutes REM-time/

REM-time in the first 2 hours of sleep (mean under 10 min.).

The two largest series with volunteers subjected to neither drugs nor experimental procedures, are those of Dement and Kleitman (1957b) with 126 nights from 33 volunteers, and Rechtschaffen and Verdone (1963) with 80 nights from 20 volunteers. The former give figures for the delay from sleep onset to first REM period as 67 min. (mean) and 45 min. (minimum), and the latter as 84 min. (mean) and 49 min. (minimum). We therefore give the value of 45 minutes as the minimum (Fig. 7) though this is lower than we have encountered in over 70 normal nights. In a cooperative study from two centres in the U.S.A., Rechtschaffen et al., (1963) have also observed very short delays of a few minutes between sleep onset and the first REM period comparable to those in Fig. 7, in narcoleptic patients (from at least some of whom amphetamine had just been withdrawn) which they state they have never before encountered in the many hundreds of other nights they have, between them, studied.

Fig. 7 shows that return to normal can take as long as 8 weeks after withdrawal. Utena et al., (1959) in Japan, where amphetamine addiction is common, made guinea pigs into meth-amphetamine addicts. Animals killed on the 15th abstinence day still showed abnormalities and probably also the animals killed on the 45th day, in that there was evidence of a decrease of aerobic and anaerobic <u>in vitro</u> glycolytic activity in studies of the animals' brain tissue.

We do not understand the basis of the abnormalities we have observed, for, notwithstanding its fundamental role in our economy/

economy, the significance of our need for sleep remains a mystery. We believe this to be the first demonstration of an abnormality both easily measurable and long-persisting (1-2 months) in any kind of human abstinence syndrome. We cannot unfortunately measure another neurophysiological abnormality, namely, that which directly underlies the most significant feature of the syndrome - the craving.

After withdrawal of amphetamine, patients describe listlessness, depression and sleepiness, but cannot easily formulate their craving - "I feel terrible, I miss them so" (Case 4), "I can't get them out of my mind, I think about them all the time" (Case 6). It is this craving that drives them to anti-social acts, to obtain their drugs without prescription from small-time traffickers and unscrupulous pharmacists, to alter, steal and forge prescriptions, and to call in rotation on different doctors from whom they conceal the addiction. Sometimes it is argued that only "psychopaths" become addicted to these drugs. In our view, many addicts would be better described as young and irresponsible (Case 2) or simply stupid (Case 4).

Case 5 had been an addict for 12 years, obtaining supplies from a pharmacist. For the last 8 years, she had often visited both her general practitioner and hospital clinics because of Raynaud's Disease, without its being realised that she was steadily consuming huge quantities of a potent vaso-constrictor agent. After withdrawal, her circulation improved enormously, her nails grew again, and she was delighted - rendering her prognosis relatively good.

Conclusions.

Therapeutic indications for amphetamine are today becoming vanishingly slight. Diet and not pills should control obesity (British Medical Journal, 1961).

Amphetamine has not shown itself to be of value in endogenous depression (Hare et al., 1962). Yoss and Daly (1961) recommend methyl phenidate as superior to amphetamine in the treatment of narcolepsy. It has been established by, among others, Kiloh and Brandon (1962), that amphetamine consumption leads to a tendency to increase the dose. Up to 10 times the "therapeutic" dose is common among amphetamine and phenmetrazine addicts. These preparations produce a detrimental effect on the individual and society. In the former, they produce an egocentricity of outlook and impairment of those skills necessary to the conduct of successful social relationships, sometimes physical harm, and occasionally a frank psychosis, (Connell, 1958; Beamish and Kiloh, 1960). In paying for the pills, addicts sometimes acquire appalling financial debts. In society, the drugs encourage shady trafficking. Persons taking these drugs certainly experience an intense craving to continue so to take them, and to obtain them by almost any means - these means, it is admitted, are not pursued to the extremes found, for instance, among morphine addicts. Extreme behaviour is unnecessary with the laxity of current controls. We have demonstrated that there can exist physical dependence.

The criteria for addictive drugs stipulated in the Report of the Interdepartmental Committee set up by the Ministry of Health/

Health and Department of Health for Scotland (1961) are therefore met by amphetamine and phenmetrazine. These drugs, and drugs with comparable actions, such as diethyl-proprion (Clein and Benady, 1962), are dangerous drugs in fact, if not yet in law.

Summary.

Evidence is presented to support the view that amphetamine and phenmetrazine preparations are truly addictive, leading to physiological dependence. Six addicts have been studied in whom easily measurable neurophysiological abnormalities appeared upon drug withdrawal.

These abnormalities affected the proportion of nocturnal sleep spent in so-called "hind-brain sleep" (with a characteristic EEG, rapid eye movements, and muscle tension changes). In the abstinence syndrome, this kind of sleep began as soon as 4 minutes (normal about 70 minutes) after sleep onset, occupied up to 70 minutes (normal about 10 minutes) in the first 2 hours and up to 48% (normal about 22%) of a whole night. Return to normal function took place immediately if the drugs were restored, but, if withheld, return to normal took 3-8 weeks.

We are grateful to Professor G. M. Carstairs for permitting us facilities. A grant from the Medical Research Council contributed to the cost of paper. We are also variously indebted to Mr. R. J. Berger, Dr. J. R. Smythies, Dr. J. D. Crombie, Dr. J. W. Affleck, Dr. Esme Simpson, Dr. P. G. Fawcett, Dr. W. D. Boyd, and Sister Falconer of the North Wing, Royal Edinburgh Hospital. We owe a particular debt to Mr. N. V. Clarke, who made an electronic device for signalling paper-jamming.

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Rechtschaffen, A., and Verdone, P. (1963), to be published.

Trethowan, W.H. (1962). Brit. med. J., 2, 188.

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Fig. 1. To illustrate the differences between the two kinds of sleep (Case 1), namely, the change from an EEG high voltage slow wave pattern, unrelaxed muscles and motionless eyes, to a low voltage EEG with relaxed muscles and jerky rapid eye movements. The muscle trace in the upper excerpt is thick because of innumerable little muscle spike potentials; in the lower trace a few only occur, particularly with respiration (the ECG is also visible). The EEG waves are all irregular except for a group of three consecutive waves of about 2.5 c/sec. visible in channels 3 and 4 just before the main eye movement burst - these precursors are the rhythmic "saw-toothed" EEG waves. Amplification was 6 mm. per 50 microvolts for the EEG and twice and a half this for the throat and eye channels respectively, time constants 0.3 sec.

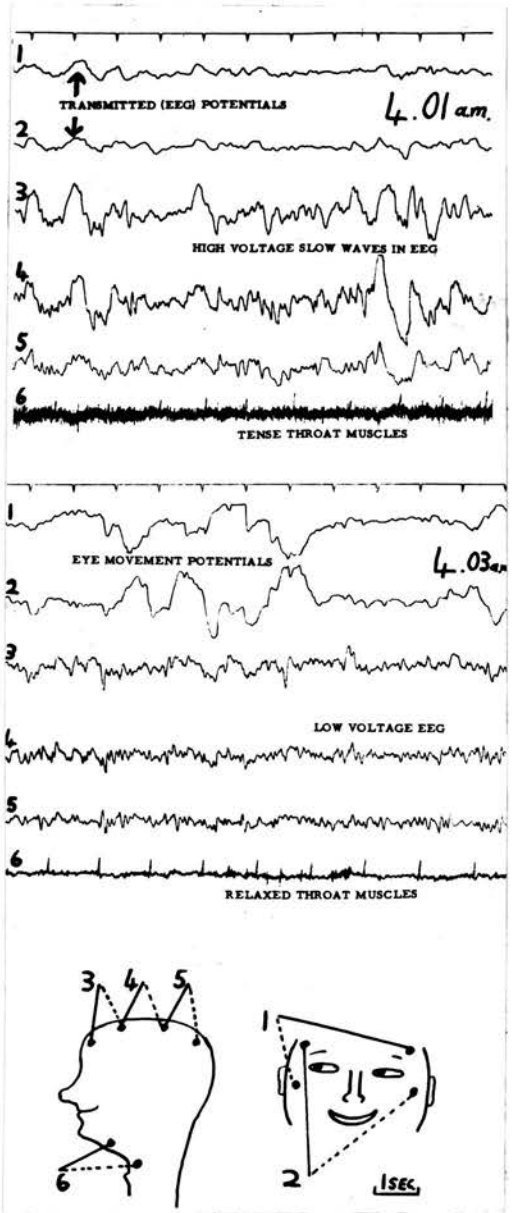
1
TRANSMITTED (EEG) POTENTIALS
4.01 a.m.
2
3
HIGH VOLTAGE SLOW WAVES IN EEG
4
5
6
TENSE THROAT MUSCLES
1
EYE MOVEMENT POTENTIALS
4.03 a.m.
2
3
LOW VOLTAGE EEG
4
5
6
RELAXED THROAT MUSCLES
3
4
5
6
1
2
1SEC.

Fig. 1.

Fig. 2. Case 1. In descending sequence on the left are blocks indicating the percentage of the whole night's sleep spent in rapid eye movement periods (REM time). The solid blocks indicate nights following days on which she received her drinamyl and the empty blocks nights following days without drugs.

On the right the number of minutes made up of rapid eye movement period sleep within the first two hours of sleep is similarly indicated.

The rise in the values whenever the drug is stopped can be seen. The values of 48% (whole night, June 15th) and 70 min. (first 2 hours, July 7th) were far above values hitherto reported or any encountered by us. The vertical time scale is not linear.

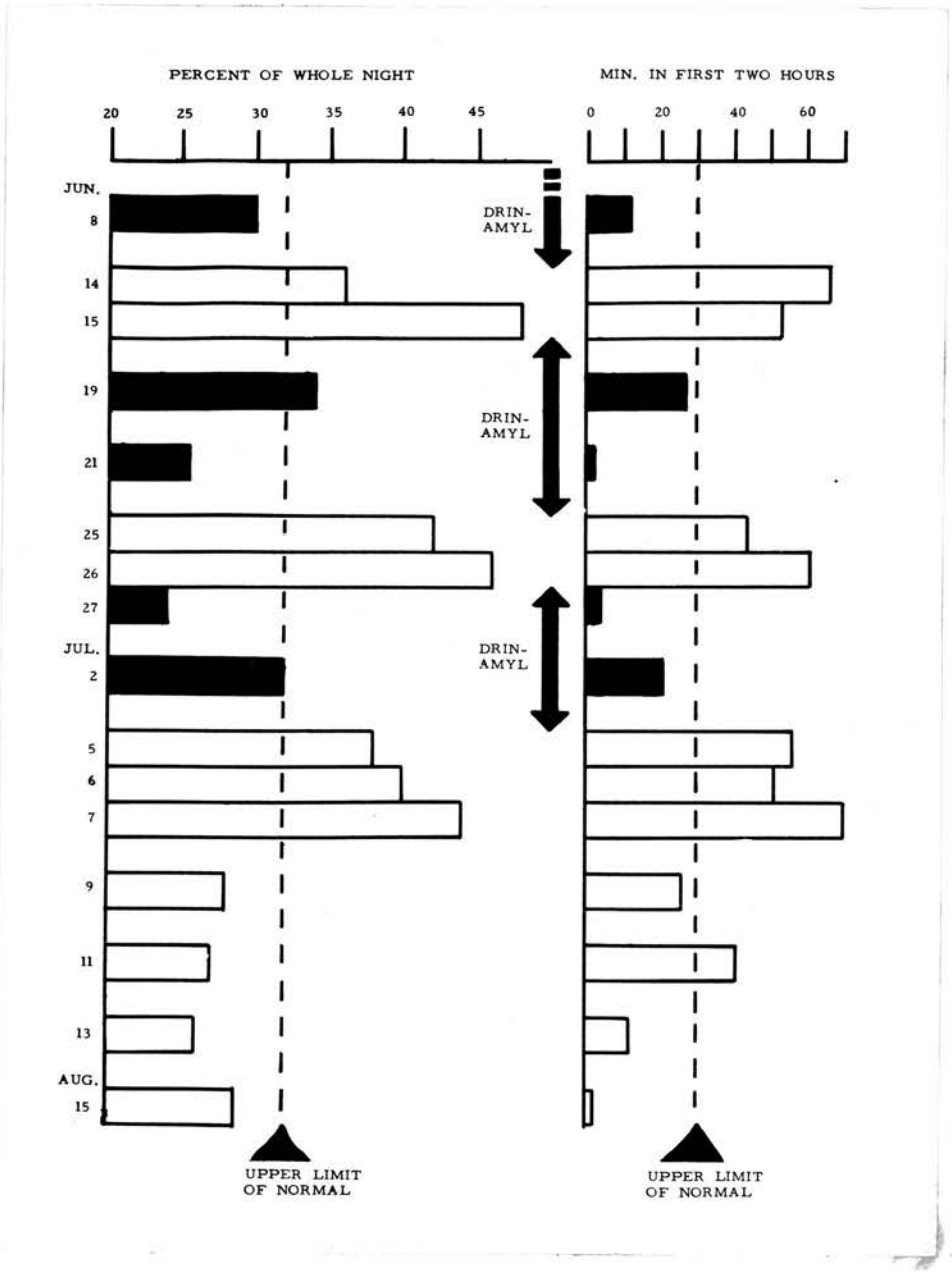
PERCENT OF WHOLE NIGHT
20
25
30
35
40
45
MIN. IN FIRST TWO HOURS
0
20
40
60
JUN.
8
14
15
19
21
25
26
27
JUL.
2
5
6
7
9
11
13
AUG.
15
DRIN-
AMYL
DRIN-
AMYL
DRIN-
AMYL
UPPER LIMIT
OF NORMAL
UPPER LIMIT
OF NORMAL

Fig. 2.

Fig. 3. Similar to Fig. 2. Case 2, only the first two hours of sleep recorded on August 16th. No rapid eye movement period within the first two hours on August 18th.

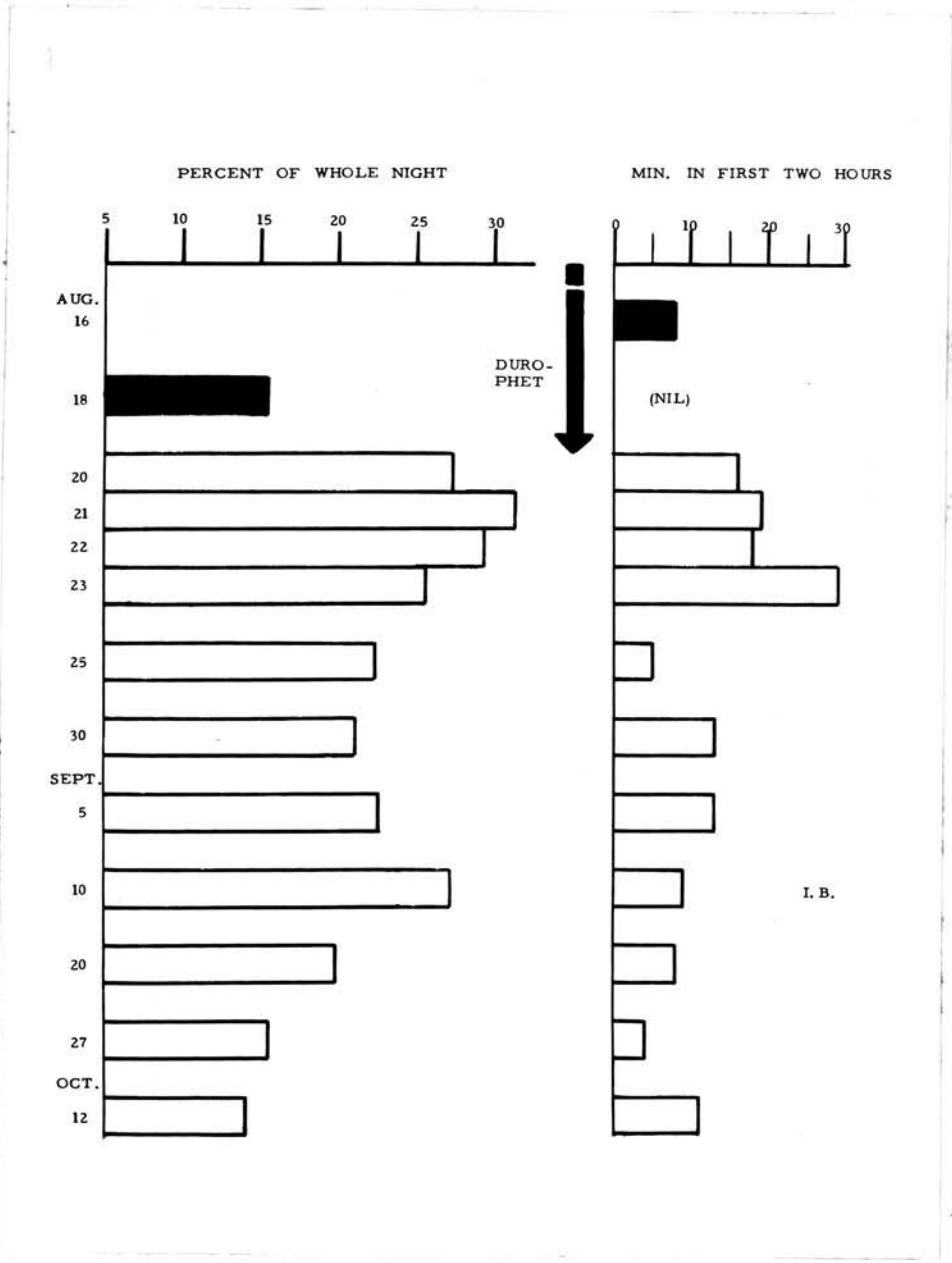
PERCENT OF WHOLE NIGHT
MIN. IN FIRST TWO HOURS
5
10
15
20
25
30
0
10
20
30
AUG.
16
18
20
21
22
23
25
30
SEPT.
5
10
20
27
OCT.
12
DURO-
PHET
(NIL)
I. B.

Fig. 3.

Fig. 4. Only the first two hours of sleep recorded on July 17th, 19th, August 2nd and 16th. Receiving only 30 mgms. per day of dexamphetamine sulphate August 11th - 19th. The slow return to a steady level within her normal range takes over a month (Case 3).

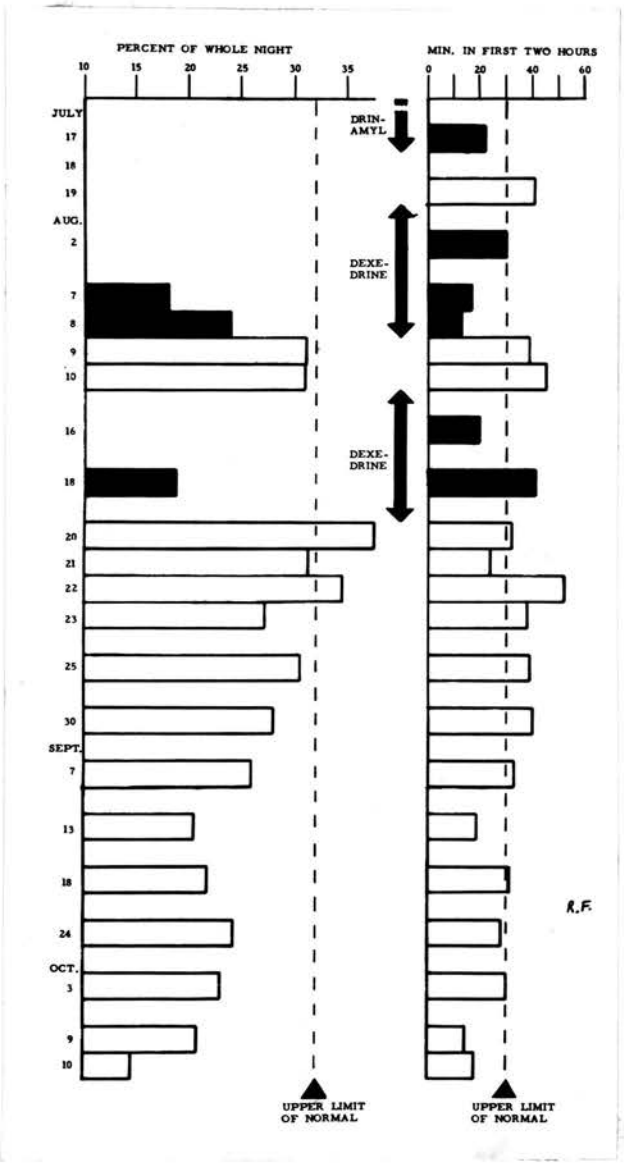
PERCENT OF WHOLE NIGHT
10
15
20
25
30
35
MIN. IN FIRST TWO HOURS
0
20
40
60
JULY
17
18
19
AUG.
2
7
8
9
10
16
18
20
21
22
23
25
30
SEPT.
7
13
18
24
OCT.
3
9
10
DRIN-AMYL
DEXE-DRINE
DEXE-DRINE
R.F.
UPPER LIMIT OF NORMAL
UPPER LIMIT OF NORMAL

Fig. 4.

Fig. 5. Showing that withdrawal of preludin (phenmetrazine hydrochloride) in a fully addicted person has the same effects as withdrawal of amphetamine (Case 4).

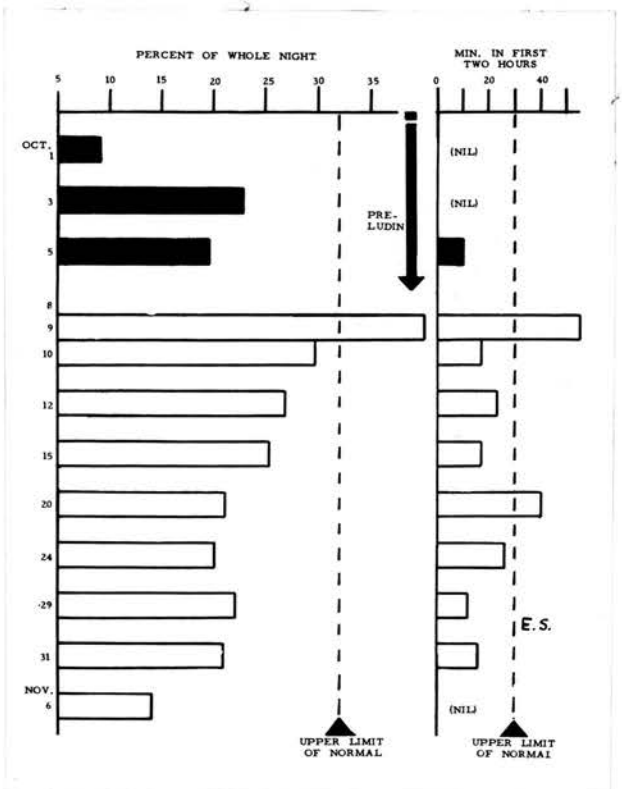
PERCENT OF WHOLE NIGHT
5
10
15
20
25
30
35
MIN. IN FIRST TWO HOURS
0
20
40
OCT. 1
3
5
8
9
10
12
15
20
24
29
31
NOV. 6
PRE-LUDIN
(NIL)
(NIL)
(NIL)
E.S.
UPPER LIMIT OF NORMAL
UPPER LIMIT OF NORMAL

Fig. 5.

Fig. 6. Case 5 shows a similar slow return to normal.

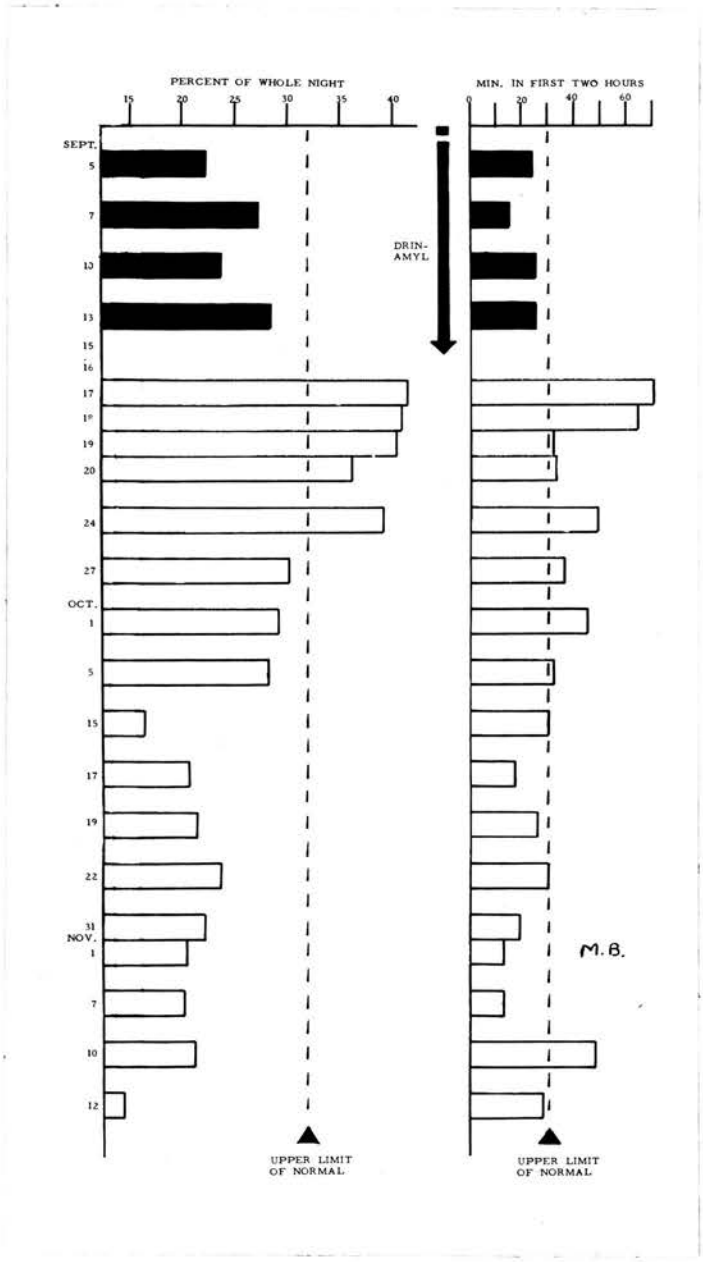
PERCENT OF WHOLE NIGHT
15
20
25
30
35
40
MIN. IN FIRST TWO HOURS
0
20
40
60
SEPT.
5
7
10
13
15
16
17
18
19
20
24
27
OCT.
1
5
15
17
19
22
31
NOV.
1
7
10
12
DRIN-
AMYL
M.B.
UPPER LIMIT
OF NORMAL
UPPER LIMIT
OF NORMAL

Fig. 6.

Fig. 7. Illustrating the occurrence of abnormally short delays before the first onset of a rapid eye movement period in the nights following withdrawal of amphetamine preparations. Pre-withdrawal means are based on six nights (Case 3) and four nights (Case 5). The first two post-withdrawal values for Case 3 are also mean values (Table I). Case 3 had one delay outside the normal range as late as the 36th day after withdrawal. Case 5, after 18 and 20 days, still had grossly abnormal values (14 min.), and not till after the 47th day did three consecutive values within normal limits occur.

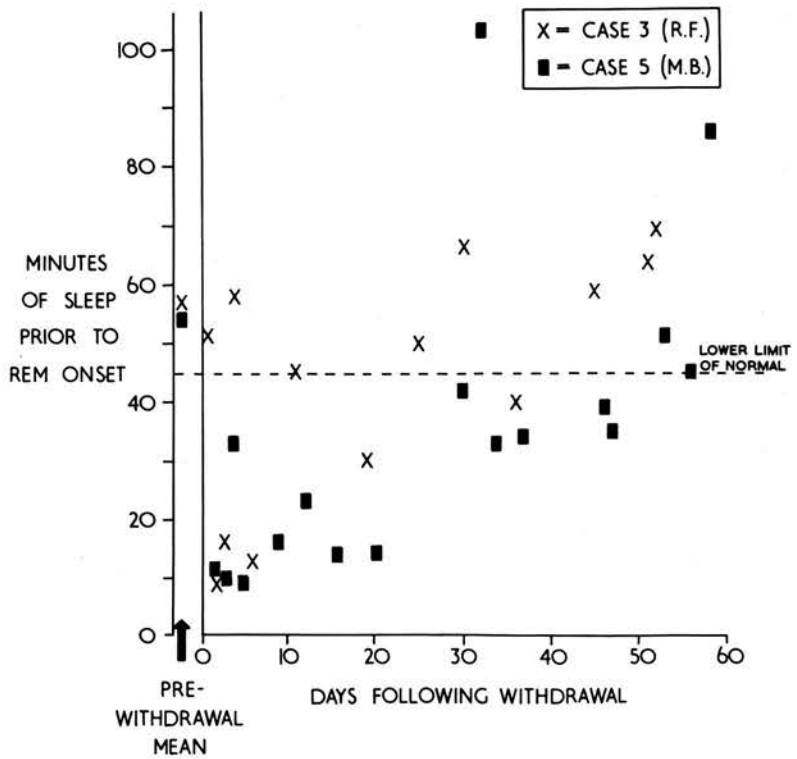
X = CASE 3 (R.F.)
■ = CASE 5 (M.B.)
MINUTES OF SLEEP PRIOR TO REM ONSET
100
80
60
40
20
0
LOWER LIMIT OF NORMAL
0
10
20
30
40
50
60
PRE-WITHDRAWAL MEAN
DAYS FOLLOWING WITHDRAWAL

Fig. 7.

A P P E N D I X.

TABLE I.

(see also Figs. 2 - 6).

TST = Total whole night sleep time (min.).
TRT = Total REM time (min.).
Delay = Delay before first onset of REMs (min.).
Dates in italics = Dates when on drugs.

Case 1.

Date	TST	TRT	Delay
Jun. 8	376	113	71
14	420	148	15
15	494	237	21
19	491	169	23
21	420	107	70
25	456	192	15
26	453	204	12
27	370	90	70
Jul. 2	463	148	79
5	443	168	46
6	480	191	37
7	435	156	37
9	493	138	61
11	444	117	39
13	501	128	42
Aug. 15	251	72	85

Case 2.

Date.	TST	TRT	Delay.
Aug. *16*			90
18	391	59	147
20	504	137	82
21	530	167	74
22	478	140	72
23	485	123	57
25	466	103	107
30	495	104	81
Sept. 5	537	121	68
10	510	139	62
20	449	89	72
27	410	63	65
Oct. 12	434	61	108

Case 3.

Date.		TST	TRT	Delay
Jul.	17			52
	19			8
Aug.	2			10
	7	254	45	61
	8	377	91	86
	9	420	130	12
	10	360	111	9
	16			61
	18	392	73	69
	20	442	166	90
	21	455	142	13
	22	432	149	16
	23	405	110	58
	25	435	133	13
	30	406	114	45
Sept.	7	392	107	30
	13	437	90	50
	18	487	106	66
	24	452	110	40
Oct.	3	433	111	58
	9	480	112	64
	10	405	58	68

Case 4.

Date.		TST	TRT	Delay
Oct.	1	379	34	154
	3	408	93	198
	5	395	78	31
	9	480	191	51
	10	468	138	55
	12	419	112	44
	15	375	95	82
	20	372	78	73
	24	336	67	85
	29	330	73	98
	31	373	78	41
Nov.	6	319	45	164

Case 5.

Date.		TST	TRT	Delay
Sept.	5	430	96	71
	7	392	107	51
	10	395	94	56
	13	397	113	38
	17	462	192	11
	18	439	180	10
	19	499	202	33
	20	407	148	9
	24	395	155	16
	27	374	113	23
Oct.	1	348	102	14
	5	340	96	14
	15	332	65	42
	17	480	100	103
	19	434	93	33
	22	411	98	34
	31	329	73	39
Nov.	1	445	91	35
	7	413	84	51
	10	482	103	45
	12	383	56	86

(19)

PHYSIOLOGY OF SLEEP ACCOMPANYING DREAMING

BY

IAN OSWALD.

(To be published in The Scientific Basis of Medicine Annual Reviews, 1962-3, University of London, Athlone Press. Based on a lecture in the British Postgraduate Medical Federation series, "The Scientific Basis of Medicine", given on February 19th, 1963).

The availability of reliable and highly sensitive ink-writing machines for electroencephalography has permitted an increased interest in many countries in the study of sleep during recent years. While asleep, we may dream, and because my title refers to dreaming, it is with human sleep especially that I shall deal.

What then does electroencephalography reveal of human sleep? As we become drowsy, the alpha rhythm seen in the waking EEG first waxes and wanes, then disappears, to be replaced by low voltage slow waves and the subsequent appearance of characteristic "sleep spindles" at about 14 c/sec., with higher voltage slow waves at 1-3 c/sec. The slow waves gradually become more and more prominent as undisturbed sleep continues. Loomis and his colleagues (1937) divided the EEG picture of sleep into stages of increasing depth, stages A to E (Fig. 1), through which the sleeper passes successively after retiring. If stimulated sufficiently, or if he stirs spontaneously, the D or E stage picture may switch briefly back to the B stage, with a subsequent gradual transition through C stage back to D or E. After perhaps 70 minutes of sleep, either without warning, or after a major movement with brief signs of arousal and then the recurrence of sleep spindles and slow waves, the picture abruptly changes to an EEG of low voltage with 2-6 c/sec. waves and a little alpha rhythm, rather like the B stage of drowsiness, and the eyes, which, during initial drowsiness, made gentle rolling movements/

movements (and which during the C, D and E stages were motionless) begin to make jerky, conjugate movements. There is also a sharp loss of tone in certain trunk muscles, and the heart rate and respiration become irregular.

After 10 minutes or so, these events are terminated, usually by a large body movement, and after a few seconds of EEG rhythms of alpha frequency (more anteriorly distributed than during wakefulness) sleep spindles soon reappear and the sequence of stages C, D and E recurs. However, at intervals of about 90 minutes during the night, there are recurrences of the low voltage EEG with rapid eye movements, persisting for some 10-50 minutes each time, and occupying in all some 20% of the night.

AMOUNT OF DREAMING.

In Chicago, Aserinsky and Kleitman (1955), who first described this recurrence of rapid eye movement periods during the night, suggested that they might be accompanied by dreaming. Dement and Kleitman (1957a) woke volunteers from sleep and on 80% of occasions got detailed dream descriptions after awakenings from sleep with rapid eye movements, but only fragmentary reports of mental content on 7% of occasions after awakenings from the other stages of sleep. Independent workers confirmed this, and reported that even those people who had believed they never dreamed would describe dreams if awakened from sleep with rapid eye movements (Goodenough et al., 1959).

It/

It has become clear that we all dream, much more than we can remember - it is our memory that is deficient. In the past, it has been proposed that patients with various forms of brain damage, including leucotomy, cease to dream. Today, such proposals would need to be supported by actual awakenings from sleep, not merely by day-time reminiscences. In our laboratory, rapid eye movement periods have been found to persist after leucotomy.

PHYSIOLOGICAL CONCOMITANTS.

A similar cyclical alternation between sleep with a high voltage slow wave EEG on the one hand, and sleep with a low voltage faster EEG and rapid eye movements on the other, has now been noted in the monkey (Weitzmann, 1961), dog (Shimazono et al., 1960), rat (Michel et al., 1961; Swisher, 1962), and rabbit (Faure, 1962), and has been especially studied in the cat by Jouvet (Jouvet et al., 1960; Jouvet, 1962), who has called the latter variety of sleep the "paradoxical" or the "hind-brain" phase of sleep (preferring now the latter to avoid confusion with Pavlov's "paradoxical phase" of inhibition). During sleep with high voltage slow waves and spindles recordable from the cortex, implanted pontine electrodes showed low voltage fast rhythms in cats. During the hind-brain sleep phase of faster cortical rhythms, slow waves and spindles were recorded from the pons, a sort of reversal having taken place. For some years, we have all been accustomed to read of the "ascending activating reticular formation", of impulses ascending within the central core/

core of the brain stem to cause cortical "activation" - a low voltage fast EEG. The impulses that ascend from the brain stem to cause the low voltage of "activated" cortical EEG appearances that accompany the hind-brain phase of sleep (as I shall hereinafter call it, though wishing we had some other agreed name) follow a different route, ascending from the so-called "limbic mid-brain" along a path through the subthalamus and septum (Fig. 2), a route mapped on histological evidence by Nauta (1958).

Jouvet brings evidence to implicate a pontine nucleus as a hind-brain sleep "releasing centre", from which impulses not only ascend but also descend to cause an invariable relaxation of skeletal muscle, most readily recordable from the cat's nuchal muscles.

The rapid eye movements of human hind-brain sleep tend to occur in clusters and, in almost two-thirds of adults, some characteristic EEG waves herald most of these clusters. Under ordinary recording conditions, these waves have so unique and immediately recognisable an appearance that Jouvet has called them "saw-toothed" waves. These waves are most marked frontally, but in some people we find they have a more widespread origin and show a phase reversal between the front and back of the head (Fig. 3).

We were looking for evidence of dream-talking when my colleague, Berger, first noticed the invariable loss of tone in muscles adjacent to the larynx with the onset of the hind-brain sleep phase (Berger, 1961). Other human muscles relax so greatly/

greatly as soon as sleep develops that, with surface electrodes, it is difficult or impossible to record a further fall with the onset of hind-brain sleep. This is even true, as Dr. R. Priest and I have found, in cases of post-encephalitic parkinsonism. Nevertheless, I have found it possible to record such a fall from the nuchal muscles that have been so much used in the cat, at times when the human sleeper has adopted some particular posture tending to cause a retention of neck muscle tone (Fig. 4). Furthermore, the saw-toothed EEG waves bear a special relation in time to the loss of human muscle tone, for, once one has become accustomed to seeing these waves one can recognise a little burst of them frequently preceding by half a minute the transition from EEG sleep spindles to the low voltage EEG with rapid eye movements, and it is in relation to such a burst of saw-toothed EEG waves that the muscle tone loss begins (Figs. 4 and 5). If one has recorded just the EEG and eye movements and is working page by page through an all-night record, suddenly one sees a burst of the saw-toothed waves and at once knows that when one turns the page the whole picture will have changed, and the rapid eye movements will have started.

TWO KINDS OF SLEEP.

When Dement and Kleitman (1957b) first gave detailed descriptions of the recurring periods of rapid eye movements, they described them as occurring during cyclical "lightenings" of sleep, and I, for one, certainly accepted the view that these were/

were light sleep periods (Oswald, 1962) - the EEG having many similarities to the B stage of drowsiness, with brief interspersions of alpha rhythm.

Animal workers have, however, called the analogous periods in the cat the phase not of light sleep but of deepest sleep, for the muscle tension and, independently, the blood pressure (Candia et al., 1962) in cats then fall to their lowest levels. In addition, the threshold for direct electrical stimulation of the ascending activating reticular formation sufficient to cause behavioural awakening is raised during this kind of sleep - it is more difficult to awaken the cats this way (Benoit and Bloch, 1960; Candia et al., 1962).

We now know the muscle tone loss to be true also of man. Furthermore, when people who have been fully addicted to amphetamine, stop taking the drug, they become extremely sleepy. You might then expect them to sleep more deeply. After such drug withdrawal (see below) they spend much more of the night in hind-brain sleep. In these two respects, we might therefore call it "deep" sleep. Yet, in this kind of sleep we can, more than in other stages of sleep, be termed "conscious", if only of our dream-world, whereas in stage E sleep with high voltage EEG slow waves we seem to approach most nearly to total oblivion. By analysis of a series of all-night records, it was found that during human hind-brain sleep, spontaneous major movements are made significantly more often than in the C, D and E stages of sleep (Oswald et al., 1963). The same is true of spontaneous rhythmic rocking movements, discussed

below/

below. Furthermore, if people are made very sleepy, not by withdrawal of amphetamine but by prolonged sleep deprivation, on the first night when allowed to sleep, they take much less hind-brain sleep and significantly more D and E stage sleep, as if the latter with its high voltage slow waves was the most needed, the most refreshing "deep" sleep (Berger and Oswald, 1962a). Skin electrical resistance rises with sleep and is reported to be greatest during hind-brain sleep (Hawkins et al., 1962). Human cortical evoked potentials following auditory stimuli (clicks) are large during the B stage of drowsiness but despite the similar EEG are very small during hind-brain sleep (Williams et al., 1962).

In the face of this conflict of evidence, it seems clear that we can only define "deep" sleep in terms of specified criteria, and that we should better recognise the existence of two kinds of sleep with no gradation between them, and which, in character discontinuous one with another, alternate during the night. Furthermore, we need both kinds of sleep, and though it is the high voltage slow wave sleep which has first priority when we retire to bed after deliberate sleep deprivation, if selectively deprived of hind-brain sleep we make up for that loss as soon as we get the chance.

The selective deprivation can be accomplished by deliberate awakening whenever the rapid eye movement period starts, as Dement (1960) first showed. He called this selective awakening a method of producing "dream-deprivation". I prefer to think of deprivation of hind-brain sleep. Alternatively, one can cause/

cause selective decrease of hind-brain sleep by prior complete deprivation of sleep (Berger and Oswald, 1962a), with, as mentioned, on the first recovery night a reduced proportion of hind-brain sleep. Following selective deprivation by either means, on subsequent nights of undisturbed sleep, there is a significant and apparently compensatory increase in the duration of hind-brain sleep. The increase, though significant, is small and does not approach that which follows amphetamine withdrawal (see below).

One can also cause a very large selective reduction in the duration of hind-brain sleep by giving barbiturates (Oswald et al., 1963). Even more potent is the same dose of barbiturate with the addition of some amphetamine (Maron and Rechtschaffen, 1963). In psychiatric practice, one encounters many persons addicted to amphetamine, amphetamine-barbiturate mixtures, and phenmetrazine (preludin). Their central nervous systems evidently become so accustomed to functioning in the presence of these drugs, which they consume in large quantities, that their sleep pattern approximates to normal. If, however, one stops their drugs abruptly, their nervous systems are temporarily unable to function normally and they spend an enormous excess of the night in hind-brain sleep, far more than has been encountered under any other circumstances (Oswald and Thacore, 1963). The abnormality subsides slowly over a matter of 3 - 8 weeks.

In normal people, the first hind-brain sleep period does not/

not occur till after at least 45 minutes of sleep, more generally 70 minutes or so, but in the addicts after drug withdrawal it begins as early as 4 minutes from the onset of first sleep spindles. Instead of occupying about 10 minutes, or at most 30 minutes, of the first 2 hours of sleep, it occupied as much as 70 minutes, and, in the whole night, as much as 48% of the total sleep duration, half as much again as the normal upper limit.

At Edinburgh, we carried out a comparison of the all-night sleep of depressed patients with insomnia and that of normal sex and age matched controls. We failed to find any close relation between the patients' recurrent periods of wakefulness and the recurrences of hind-brain sleep. Awakening, though perhaps occurring especially towards the end of a rapid eye movement period, on the whole occurred irregularly. Even when a patient slept very little, some was taken as hind-brain sleep (Oswald et al., 1963).

In a further controlled study, with Drs. Berger and Thacore, we failed to find any effect upon the nocturnal sleep pattern of large doses of oral l-tryptophan.*

I should like, if I may, to use these last two investigations to illustrate certain techniques needed when using the EEG. All EEG interpretation is subjective and dependent on experience. So, in these investigations, after all the EEG night-records had been accumulated, they were each assigned a code number by a colleague and presented to me so that, when making judgements of each change of EEG pattern, I could not know whether I had before me the EEG of/

x

FOOTNOTE.

Further experience shows that large oral doses on retiring can cause an abnormally early onset of the first period of "hind-brain" sleep - within a few minutes of the first appearance of sleep spindles. This action is comparable to that observed with amphetamine withdrawal. If confirmed, it will be the first evidence of a substance, known to be involved in natural CNS metabolism, capable of modifying sleep.

of a patient or of a control. Having to work through miles of paper at the end like this is tedious, but essential. Whenever one reads claims that a particularly high proportion of abnormal EEGs are to be found among, say, criminals, boxers, or patients with some physical disorder, it is as well to be reserved until one is assured not only that the age distribution was the same as in the control group, that the patients and controls were done under similar conditions during the same period of time, by the same recordist using the same machine, but that whoever reported on the EEGs could not know whether any given record was from the experimental or the control group. In research in this field and in others, particularly psychiatry, there is an especial need for safeguards and techniques of the kind used by experimental psychologists.

SIGNIFICANCE OF THE RAPID EYE MOVEMENTS.

Kleitman, Dement, and their colleagues propose that the rapid conjugate eye movements of hind-brain sleep are "scanning" movements, in which the dreamer looks around at the visual events of his dream. We were able to confirm one of their claims (Dement and Wolpert, 1958; Berger and Oswald, 1962b).

My colleague Berger woke volunteers 103 times from rapid eye movement periods and tape recorded 89 dream narratives. I was never there at night during that series. Subsequently, I classified each dream narrative as "active" or "passive" according to whether the events described, had they occurred in real life, were/

were likely to have been accompanied by much or little in the way of eye movements. Later, he gave me the 89 eye movement records which had preceded each awakening and dream report, each having been assigned a code number. These too I classified as "active" or "passive", depending on the amount of eye movements. The code was then broken and (to my own surprise) there was a very close relation between "active" dreams and many eye movements, and between "passive" dreams and few eye movements. Fifty dreams had been judged "active" and in no less than 42 instances the corresponding eye movement records had been classed as "active". Of the 39 dreams judged "passive", however, 23 had been tape recorded after "passive" eye movement periods ($P < .001$).

Additionally, we took records from another series in which on some nights subjects received barbiturates and on other nights dummy tablets. By a similar "blind" technique - and in this case, actually counting the number of what I judged to be individual eye movements in matched 5 minute samples - we found barbiturates to reduce the amount of eye movement "activity" within an on-going period of hind-brain sleep (Oswald et al., 1963). Whether this is related to less "active" dream fantasies with barbiturates we do not know.

It could be argued that some general "invigorating" influence causes many eye movements and, quite independently, "active" dreams. It is particularly difficult to reconcile a "scanning" hypothesis with the report that not only are the rapid eye movements present in cats deprived of their neocortex but also in human patients/

patients believed on clinical grounds to be decorticate (Jouvet et al., 1961). However, American workers go further and report close agreement between the direction of the last few eye movements prior to awakening, and predictions of the probable last few eye movements judged on the basis of the dreamers' reports (Roffwarg et al., 1962). The degree of closeness of agreement was a subjective judgement by the experimenters ("good", "fair" and "poor").

Their proposal may be finally established when groups, each consisting of, say, 5 of the records of eye movements, previously matched for equal degree of "activity", are given with the relevant dream reports and predictions made therefrom to an entirely independent judge asked to guess which eye movement period went with which dream report. In such a case, a 20% success rate would be expected by chance alone and a statistical evaluation of the judge's performance would be possible.

Supporting the American claim are our own findings with blind men (Berger et al., 1962). I had expected congenitally blind men to have rapid eye movements in their sleep, just like normal people, but was wrong; 3 such men we studied did not. Yet men blind only a few years who still had visual experiences in their dreams still had rapid eye movements. We had two other men who had once seen but who had been blind 30 and 42 years respectively, who said they no longer visualised things either awake or in their dreams, and they had no rapid eye movements in their sleep, though they/

they had the usual recurrent periods of low voltage EEG and dreamed (of fantasy adventures lacking visual characteristics).

Also relevant is the report by Evarts (1962) of the rate of discharge of single neurones in the cat visual cortex. The rate was low in sleep with high voltage EEG slow waves and similar to that of wakefulness with closed eyes, but the rate of discharge was high during hind-brain sleep, especially at times of rapid eye movement bursts, and similar in rate to that of wakefulness with visual searching.

STIMULATION DURING HUMAN SLEEP.

Evarts is among those who have regarded hind-brain sleep as the deepest sleep because of the difficulty in provoking wakefulness by electrical stimulation of the reticular formation. This is really an unnatural form of stimulation, of a kind not encountered in evolution. I believe that, in nature, the cortex regulates awakening upon external stimulation. I believe too that human studies of stimulation in sleep, because of man's learning in terms of symbols and his power of recall, can reveal much that is impossible in animal experiments. Thus, using natural and physiological stimulation, it was possible to show that the cerebral cortex still functions discriminatively during sleep with EEG slow waves and spindles (Oswald et al., 1960). For instance, when we played a tape recording consisting of spoken names occurring alternately with spoken names played backwards, EEG responses occurred significantly more often to the meaningful stimuli/

stimuli than to the physically identical but meaningless stimuli. The same was true of galvanic skin responses (Fig. 6).

Such complex auditory discriminations between speech sounds must, it is believed from other animal and clinical evidence, depend upon cortical action, subsequent to which impulses must have passed to the brain stem to promote the subsequent responses.

In more recent experiments, Berger (1963) got volunteers to fall asleep at night against a background of "white" noise (a sort of rushing and roaring sound) of varying volume. During each rapid eye movement period, he also played a tape-recorded voice speaking a name repeatedly at intervals of a few seconds. The name varied from one rapid eye movement period to the next, but only four names were used for each volunteer, spread over several nights. After some minutes of stimulation, he would wake them and tape record their dream reports. If they had been cats, this could not have been done. But it was possible to show that simply because awakening does not occur upon stimulation, it does not mean that the organism is not responding during hind-brain sleep. The meaningful stimuli were incorporated into the dream-life. They did not cause awakening; they were altered in form and significance, presumably through cerebral cortical action. In order to assess this objectively, when Berger had 89 dream narratives, he gave them to me. I was told the four names that had been played during the sleep of each subject but not <u>which</u> of the four had been played during any particular rapid eye movement period, and was required to guess which one it was. It proved/

proved possible to judge correctly not once in four times, as would be expected by chance, but on 32 of the 89 occasions, the probability of doing so well by chance alone being less than one in 200.

The transformations in the dreams were fascinating. The man stimulated with "Gillian", the name of an ex-girl friend, described the entry halfway through his dream report, of an old woman "who came from Chile" (she was a Chilean - Gillian - an old woman - an ex-girl friend). The man stimulated with "Sheila" described how he had "left a copy of Schiller there" - the German poet. A man stimulated with "Naomi" began his report thus: "We were travelling up north, having an aim to ski. My friend, he said, 'Oh'". Naomi - "anaimtoski" + "Oh" - Naomi.

The presence of dreaming during human hind-brain sleep may be considered established. But is it confined to that phase of sleep? There is increasing evidence that some form of mental life can go on in all stages of sleep. It has been shown that galvanic skin responses follow meaningful words in stage D sleep with spindles, suggestive of some sort of "mental" life (Oswald et al., 1960). Foulkes (1962), when waking people from their sleep, did not ask, "Have you been dreaming?", but, "Was anything passing through your mind?". He got an affirmative reply almost as often when waking from the EEG sleep spindle stages as when wakening from hind-brain sleep. There were, however, important qualitative differences. In the former case (the slow wave and spindle wakenings), "thinking" of, for instance, recent events was often/

often reported, whereas after awakening from hind-brain sleep, "dreams" were often reported - adventures far in time and place from the laboratory bed, with much imagery and strong emotion, of hostility and the like. These special characteristics of dream-thinking, with their elaboration and the oblique play on words (like the Gillian - Chile quoted), are very interesting. Only in our dreams can we leave behind reality and have experiences like those of the acute schizophrenic patient - as many have pointed out, and one wonders whether it is purely chance that amphetamine should have so special an action upon this mode of cerebral function, for it is above all others the substance capable of causing a psychosis clinically indistinguishable from acute schizophrenia.

Further evidence of mental life in other stages of sleep is to be found in sleep-talking. Sometimes this occurs during a hind-brain sleep period, but most often in association with a major movement interrupting sleep spindles and a high voltage slow wave EEG, suggesting that the talking might bear some relation to immediately preceding mental content (Fig. 6).

There is another source of evidence which I find of especial interest, namely, a class of sleep disorders of which sleepwalking is the best known example. There are wartime descriptions of sleeping soldiers rising from their beds, shouting and rushing around as if again in battle (Pai, 1946). They were "living" in their dreams. But I do not know of any electrophysiological studies of sleepwalking,

Easier/

Easier to study is another disorder involving also a major degree of motor activity - the condition known as jactatio capitis nocturna, that is to say, rhythmic banging or rolling of the head and body during sleep (Putnam-Jacobi, 1880; Zappert, 1905).

If a baby monkey is separated from its mother, in its state of loneliness and fear it seems to comfort itself by crouching and making rhythmic to and fro rocking motions (Harlow and Zimmermann, 1959). Human infants of about a year often do the same when put to bed. In non-institutionalised older children, it is not common by day, but in a few these rhythmic movements persist into later life, occurring either on retiring to bed and/or actually during sleep (Lourie, 1949; Evans, 1961).

Now any form of rhythmic activity or stimulation, particularly if it is violent, tends to produce a state of calm, relaxation and sleep. Thus normal adults with their eyes glued open, when stimulated by rhythmic electric shocks causing movement and sharp sensations, coupled with intense flashing lights and rhythmic jazz music - all synchronised in their rhythm, rapidly pass into sleep (Oswald, 1960).

This is a primitive response found throughout the animal kingdom, especially in young creatures. It offers, if you like, an escape from harsh reality into slumbrous tranquillity. Rhythmic rocking is a comfort habit (as is thumb-sucking). It soothes not only the babe in arms, but the old lady in her rocking chair.

I/

I have recorded a series of nights with two males, aged 20 and 13. Both are physically normal and neither has any frank psychological abnormalities by day. Both have engaged in rocking during their sleep since infancy. The first sleep-walked till aged 12; the death of his mother and ill-treatment by mother-substitutes between his 5th and 7th years probably contributed.

I have never witnessed a more remarkable spectacle than that presented by these head-bangers. The sleeping 20-year old, lying to his right, would suddenly and violently fling his head and body rhythmically to left and (more vigorously) to right. The 13-year old would abruptly turn onto his hands and knees, and flexing and extending at the knees and hips would hurl his head rhythmically like a battering ram at the pillow and bed head. These movements they made at approximately one per second (which is an extremely strenuous rate of working).

I recorded 50 rocking episodes from the older and 4 from the younger of these two. Of the 50, two-thirds arose abruptly from sleep without previous movement. They were most frequent and most violent during hind-brain sleep (with inferred dreaming), and occurred during it, in terms of unit time, roughly 4 and 10 times as often as in the EEG C stage and the EEG D and E stages respectively. Whereas ordinary turning-over movements were followed, as is normal, by a brief period of EEG rhythm of alpha frequency and by a brief tachycardia, this was not so after the rocking movements (Fig. 7.).

More/

More interesting to me was the abrupt onset of rocking during sleep with high voltage EEG slow waves and spindles (Fig. 8). The 13-year old's head-banging also interrupted sleep with EEG spindles and slow waves. In his case, perhaps not surprisingly in view of the more complex initial movements required for getting onto his hands and knees, rhythms of alpha frequency appeared (Fig. 9), whereas in the older man EEG waves of only 6-7 c/sec. could sometimes be seen during the movements.

I regard this rocking as a pattern of behaviour which is motivated in nature and cannot but wonder whether it is occurring in sleep because of some concurrent unhappy thoughts of a kind from which the individual long ago learned to seek relief in rhythmic activity.

It does seem then as if some kind of mental life may be possible at any stage of sleep. Dreaming must not be defined in terms of some particular kind of sleep, with or without rapid eye movements. Dreaming is living in an unreal, fantasy world.

"THAT KNITS UP THE RAVELL'D SLEAVE OF CARE".

Sleep is so fundamental to our economy that most of us take it for granted. If we asked a man which basic need - let us say for food, for sex, or for sleep - he would least readily forgo for a whole week, I think there would be little doubt which he would name. We need sleep. If deprived of it, after about 60 hours most people begin to see surfaces of objects changing, many have brief hallucinations - seeing people looking at them who/

who vanish as they approach, or hearing a dog bark or a voice speak (Morris et al., 1960; Berger and Oswald, 1962a). Some develop a psychosis with systematic paranoid delusions, persisting generally till they are allowed to sleep. It is as if they were dreaming while up and about with eyes open.

Though we need sleep, yet we know little about it - what it is that causes us to be overwhelmingly sleepy if deprived of it for a few nights, why we feel so much better -especially if physically ill - after it. When we understand this better, we shall have acquired knowledge of one of the fundamental mechanisms of the living brain.

ACKNOWLEGMENTS.

I wish to acknowledge my debt to my colleague, Dr. R. J. Berger. Dr. J. W. Affleck and Dr. Margaret Methven kindly allowed me to study the head-bangers, and the Medical Research Council provided a grant towards the cost of some of the work described.

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Fig. 1. The characteristics of the various stages of sleep. 1145 hr. - Stage A or drowsiness, with the alpha rhythm, best seen in channel 3, waxing and waning, and the rolling eye movements revealed in channels 4 and 5. 1146 hr. - Stage B, the alpha rhythm now lost and low voltage slow waves but no spindles present in the EEG; the eyes continue to drift slowly. 1158 hr. - Stage C, "sleep spindles" at about 14 c/sec. and most prominent anteriorly are present together with large slow wave complexes; the eyes no longer move, only transmitted EEG potentials being revealed by channels 4 and 5. 0008 hr. - Stage D with the sleep spindles now slightly slower in frequency and a general slowing and increase of voltage of the background EEG. 0041 hr. - Stage E, the EEG being dominated by high voltage waves at about 1 c/sec. Note that channel 6 which has a thick appearance owing to innumerable muscle spikes changes little between these stages.

The EEG at 0052 hr. is similar to Stage B, though with a little alpha rhythm in channel 3 and "saw-toothed" waves (see Fig. 3) preceding the rapid jerky eye movements revealed in channels 4 and 5. The muscle tonus over the front of the neck is greatly diminished (channel 6) and the heart-rate irregular (channel 7). This is an excerpt from the "hind-brain" (or "paradoxical") phase of sleep.

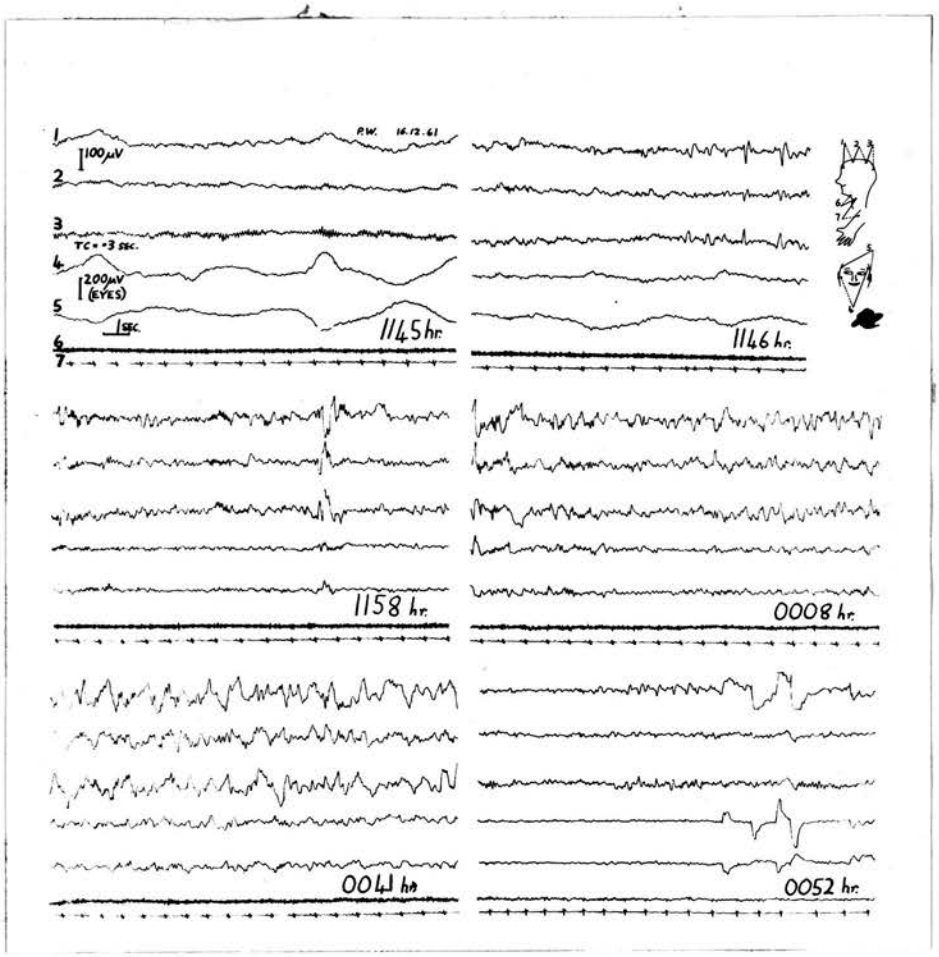

P.W. 16.12.61
100μV
TC = 3 sec.
200μV (EYES)
1 sec.
1145 hr.
1146 hr.
1158 hr.
0008 hr.
0041 hr.
0052 hr.

Fig. 1.

Fig. 2. Brain stem of cat. Lesions 1, 2, 5, and 6 prevent cortical EEG activation (i.e. the low voltage rhythms) during the "hind-brain" phase of sleep but do not prevent cortical EEG activation in the case of ordinary arousal. The low voltage EEG rhythm during the "hind-brain" phase of sleep more closely resembles those of wakefulness than is the case in man.

Lesions 3 and 4 prevent arousal but permit the cortical EEG activation of the "hind-brain" sleep phase.

The black path ascends from the "limbic mid-brain" (after Nauta, 1958).

8 = nucleus pontis caudalis ("releasing centre" for "hind-brain sleep").

(Reproduced with permission from Jouvet (1962)).

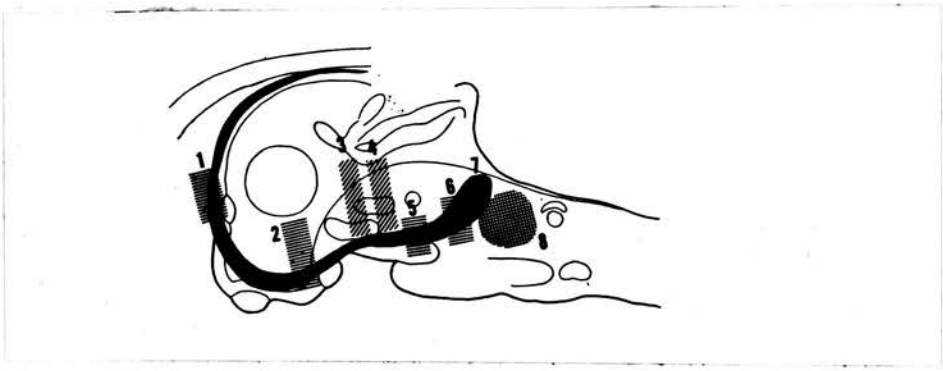


Fig. 2.

Fig. 3. Example of bursts of "saw-toothed" EEG waves heralding clusters of rapid eye movements. It can be seen in channels 1 and 3 that these waves show a phase reversal between the front and back of the head. In addition, in one segment (P.W.), a few fast muscle spikes are visible, indicating the little facial twitches that are sometimes associated with rapid eye movements. The appearance of the saw-toothed waves in channel 1 should be noted; they tend to have a rapid up-going and more gradual down-going component (see Figs. 4 and 5 also).

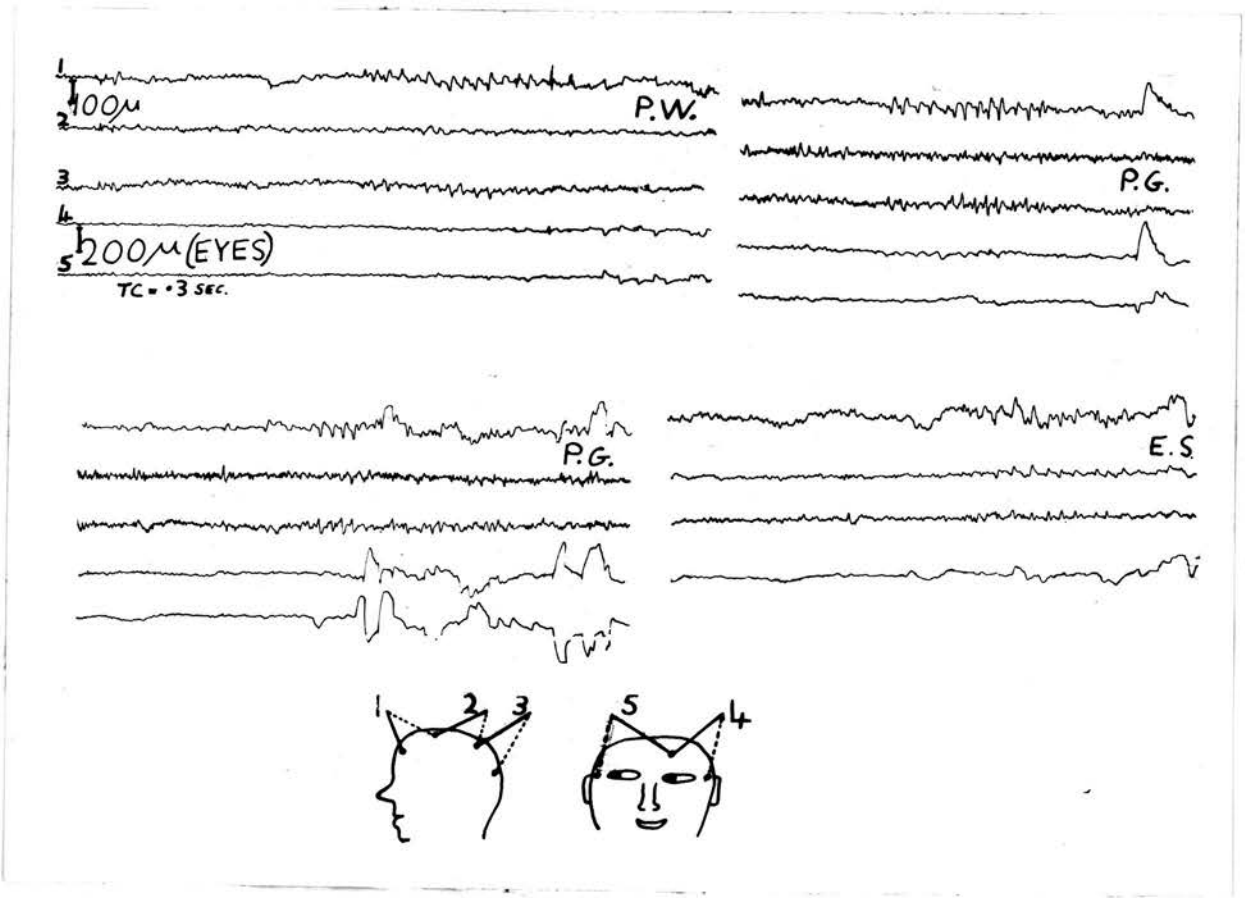


Fig. 3.

Fig. 4. The four segments of record are continuous with each other and show the transition from sleep with high voltage slow wave complexes and spindles to "hind-brain" sleep with rapid eye movements. The associated loss of muscle tension in the nuchal muscles is obvious (the EKG shows as well).

Note especially that, in the topmost segment, the steady muscle tone is momentarily lost concurrently with a rhythmic burst of EEG waves of which two (channel 1) have the same shape as the "saw-toothed" waves of Fig. 3. Irregular EEG slow waves and spindle activity recurs and the muscle tone remains considerable again, then declining once more and largely absent for half a minute before the first eye movement (on extreme right of third segment). A vigorous cluster of eye movements can be seen in the middle of the fourth segment in which the absence of EEG sleep spindles should be noted. The EEG is of higher voltage in this 13-year old boy than is usual in adults.

Channels 3 and 4 were recorded at half the amplification of channels 1 and 2, and channel 5 at four times the amplification. Time constants 0.3 sec.

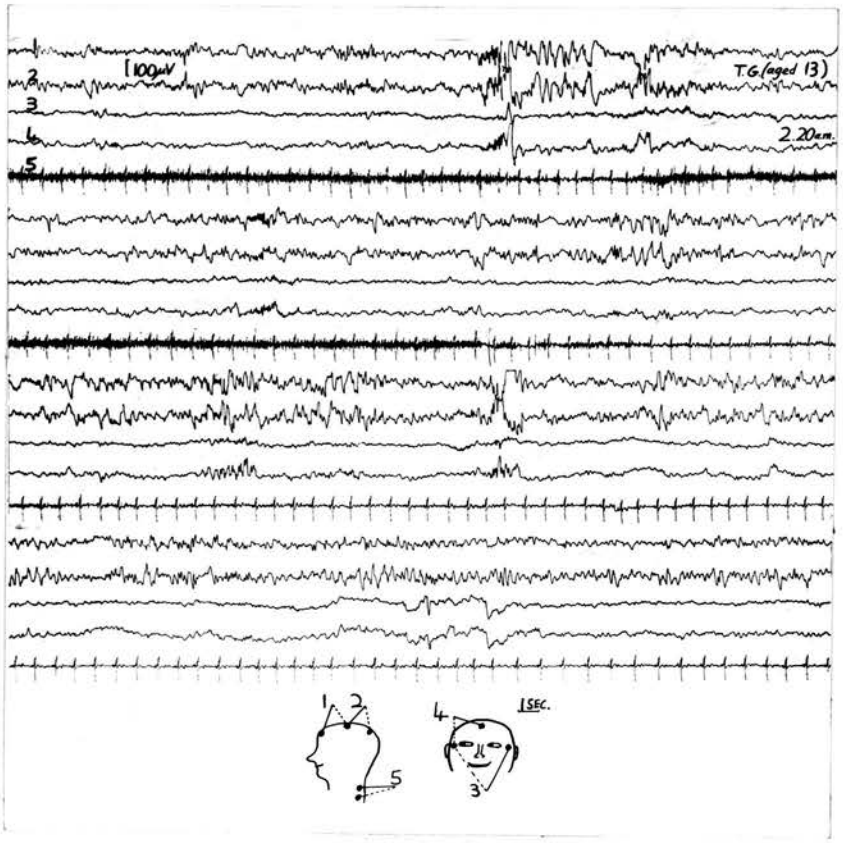


Fig. 4.

Fig. 5. The decline of muscle tension over the front of the neck in an adult with the transition from sleep with high voltage slow waves and spindles in the EEG to "hind-brain" sleep with rapid eye movements. The five segments are continuous with each other. In the second segment can be seen a momentary loss of muscle tone concurrent with a group of EEG rhythms which contain, in channel 1, two adjacent waves having the "saw-toothed" appearance when muscular relaxation is maximal. EEG slow waves and spindles recur but on the right of the fourth segment a group of "saw-toothed" waves accompany loss of muscle tone. Again spindle activity is present with return of some muscle tone (on left of fifth segment) but as "saw-toothed" waves herald the first rapid eye movements muscle tone decreases again. Amplification of channel 6 is four times that of the EEG channels.

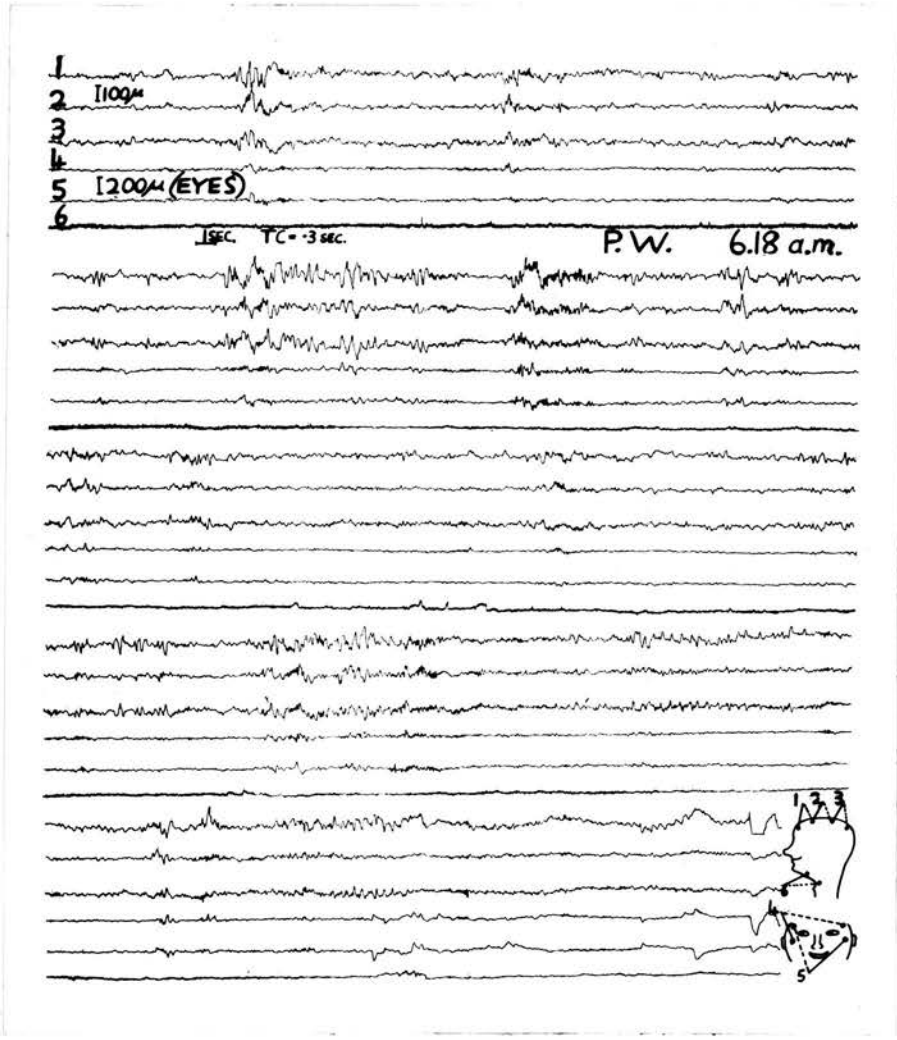


Fig. 5.

Fig. 6. Two examples of sleep-talking. The two topmost segments of record are continuous with each other, as are the three bottom segments. Female, aged 67.

The upper example shows the most common occasion of sleep-talking - a few incoherent words accompanying a brief major spontaneous movement interrupting sleep with high voltage slow waves and spindles which become quickly re-established. It is a matter for speculation whether some immediately prior mental content was related to the talking.

The lower example shows an incident of major movement and, soon after, talking, in the course of a period of "hind-brain" sleep with rapid eye movements and inferred dreaming. Note the alpha rhythm at the time of talking.

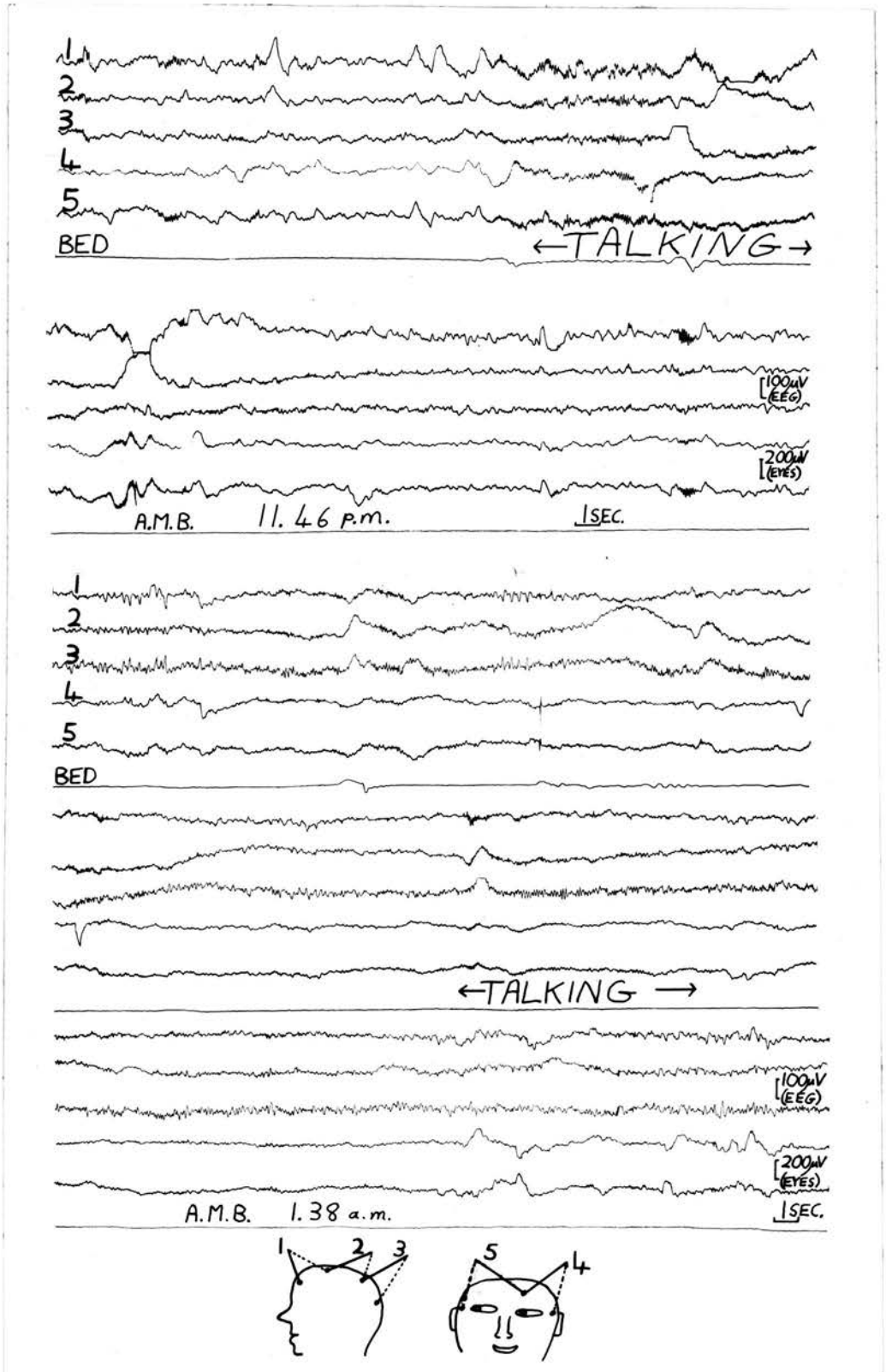

1
2
3
4
5
BED
←TALKING→
100μV (EEG)
200μV (EYES)
A.M.B. 11.46 P.M.
1 SEC.
1
2
3
4
5
BED
←TALKING →
100μV (EEG)
200μV (EYES)
A.M.B. 1.38 a.m.
1 SEC.
1 2 3
5 4

Fig. 6.

Fig. 7. Electrode positions as for Fig. 8. The upper two excerpts show rocking episodes occurring during rapid eye movement (REM) periods of sleep. In the top excerpt, no alpha rhythms are provoked and the EEG rapidly returns to the low voltage slow pattern, with rapid eye movements, the heart-rate showing, if anything, a slight fall. In the next excerpt, only the start and end of an 82-second period of continuous rocking are shown. Note the diminution of vigour by the end and the brief appearance of 7 c/sec. EEG waves anteriorly. Despite the prolonged vigorous exercise, the heart-rate after movement remains slow.

The bottom excerpt from the same patient illustrates, for comparison, a typical major body movement, such as all people have in the night. It is similar in duration to the upper rocking episode. Alpha rhythms follow for about 8 seconds till slower waves return. The heart-rate rises sharply for several seconds after the movement. Paper speed 1.5 cm./sec. and time constants of 0.3 seconds. Marks have been added to indicate more clearly the heart-rate.

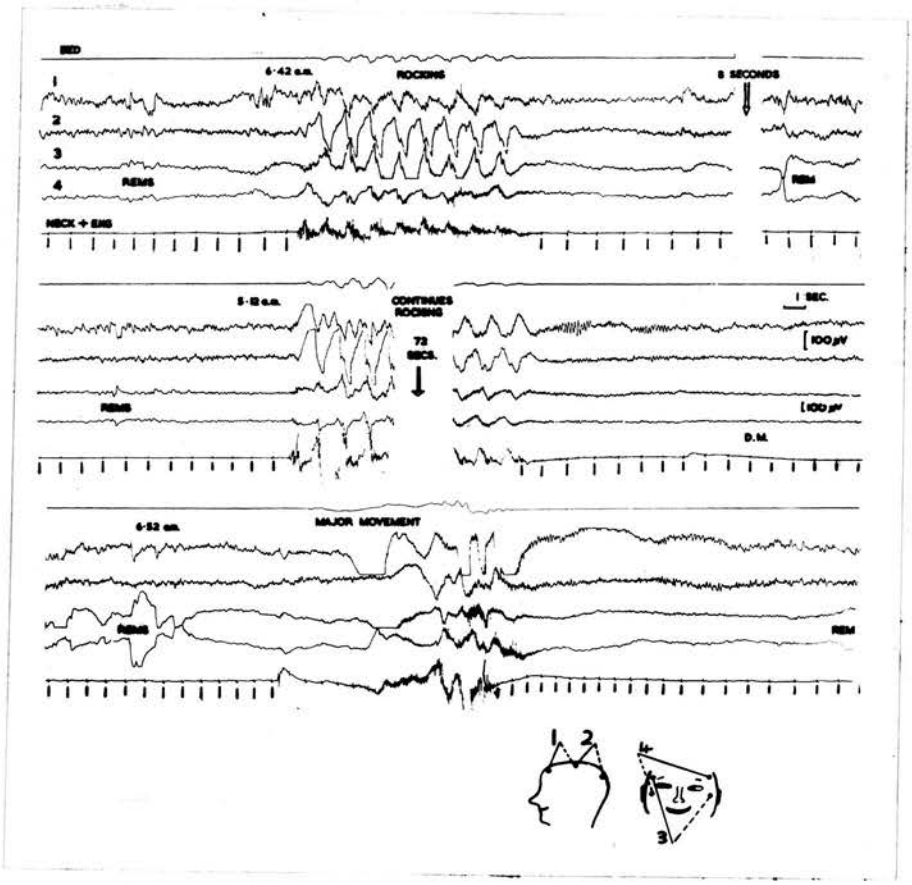
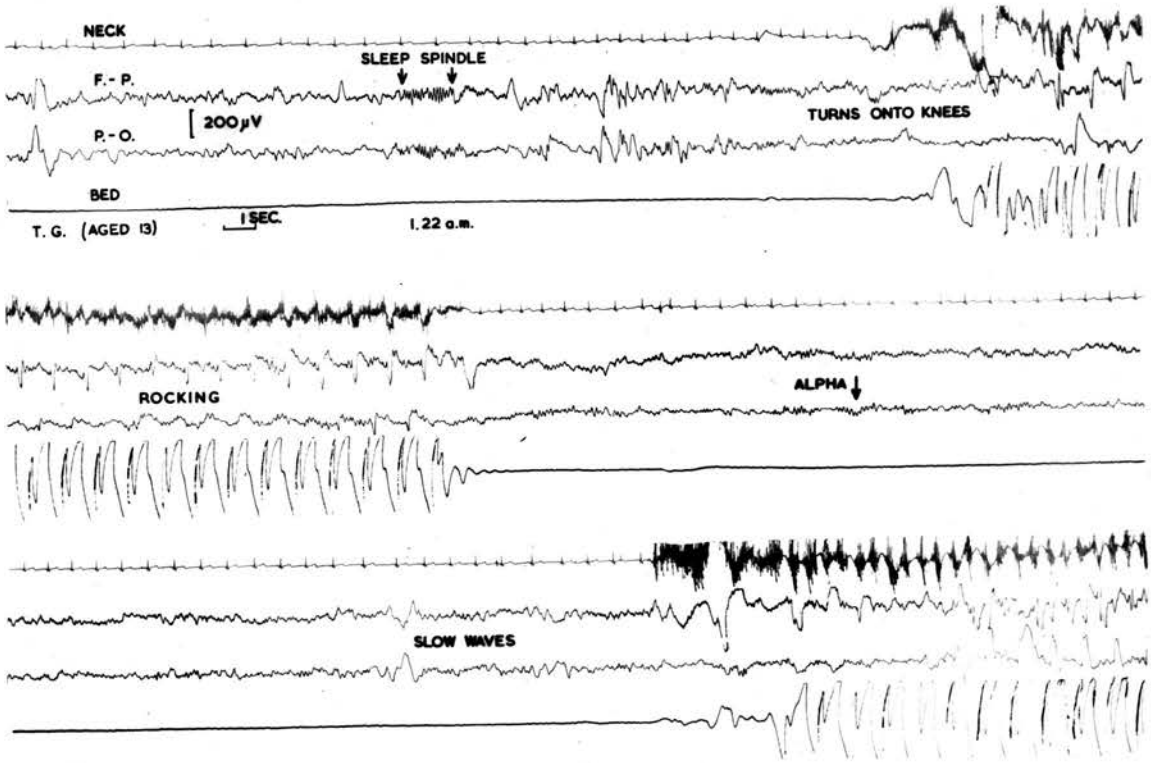

BED
6·42 a.m.
ROCKING
8 SECONDS
1
2
3
4
REMS
REM
NECK + EMG
5·12 a.m.
CONTINUES ROCKING
72 SECS.
1 SEC.
100 μV
100 μV
REMS
D.M.
6·52 a.m.
MAJOR MOVEMENT
REMS
REM
1
2
3
4

Fig. 7.

(see 3 pages on)

Fig. 8. Showing an episode of rocking during sleep. The EEG signs are of stage D sleep with anterior 14 c/sec. sleep spindles (channel 1) occurring up to the moment of onset of rocking, and slow waves at about 1 c/sec. (channels 3 and 4). Large slow waves appear to continue despite the vigorous rocking, but about 10 seconds later the EEG takes on a low voltage appearance for about 20 seconds, whereafter spindles and large slow waves reappear.

The time constants of the EEG machine had been reduced to .03 sec. in channels 1 and 2. This had been done in an attempt to eliminate slow movement artefacts during rocking, in the hope of examining the EEG waves better. However, this manoeuvre much attenuated the slower EEG waves in channels 1 and 2. The EEG slow waves can, however, be seen in channels 3 and 4, which, though primarily for eye movement potentials, show EEG potentials as well. Channels 3 and 4 are at half the amplification of channels 1 and 2. The "NECK" channel derived from an electrode over each sternomastoid muscle (and was at low amplification).


NECK
SLEEP SPINDLE
F.-P.
200μV
P.-O.
TURNS ONTO KNEES
BED
T. G. (AGED 13)
1 SEC.
1.22 a.m.
ROCKING
ALPHA
SLOW WAVES

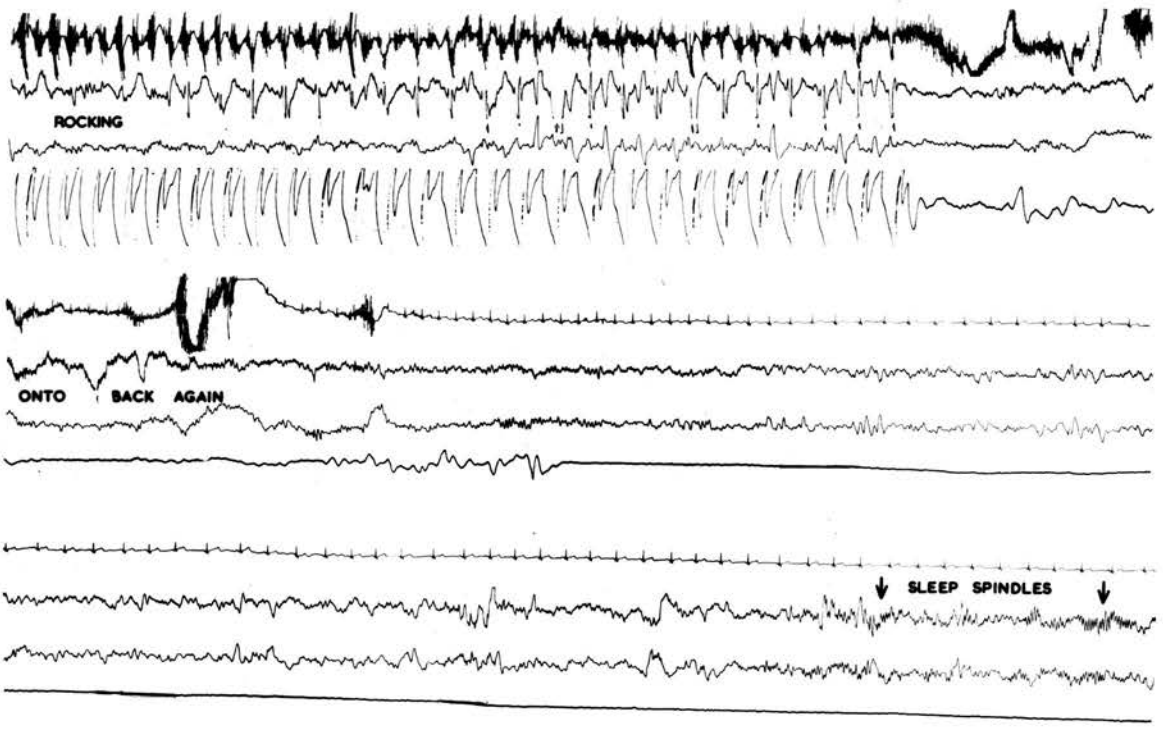

ROCKING
ONTO
BACK AGAIN
SLEEP SPINDLES

Fig. 9.

Fig. 9. The EEG is derived from midline fronto-parietal (F.-P.) and parieto-occipital (P.-O.) electrodes, with additional electrodes over the nuchal muscles (NECK).

The six excerpts are continuous with one another. Note the abrupt onset of to and fro head-banging from sleep with high voltage EEG slow waves and 12 c/sec. sleep spindles most marked anteriorly. Alpha rhythm, most marked posteriorly, and mixed with low voltage slow waves, can be seen during the latter part of the first rocking episode. Larger slow waves quickly return but another period of rocking begins, ending with irregular movements as he turns onto his back again. EEG slow waves reappear as movement ceases, and sleep spindles after a further 40 seconds. Paper speed 1.5 cm./sec. and time constants of 0.3 seconds. Low amplification of "NECK" channel.

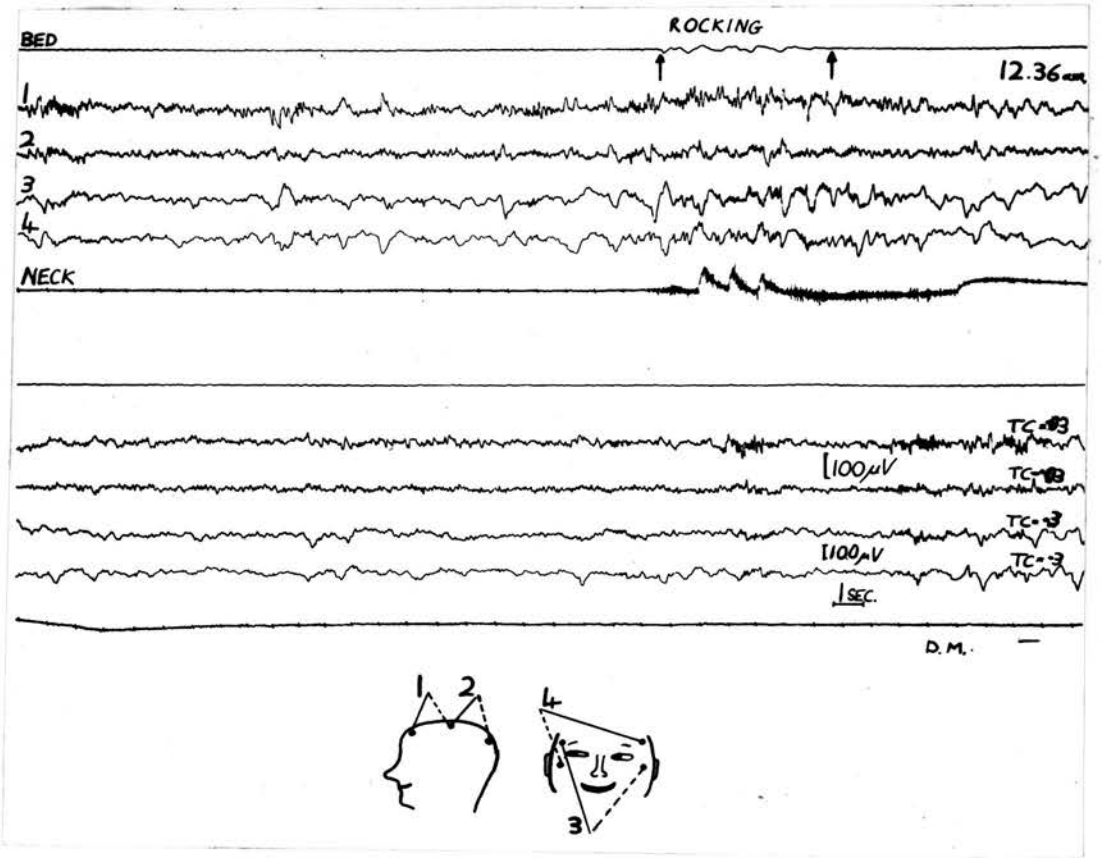

ROCKING
BED
12.36
1
2
3
4
NECK
TC=3
100μV
TC=3
TC=3
100μV
TC=3
1 SEC.
D.M.
1
2
4
3

Fig. 8.